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The Classification Of The Schizophrenias According To Symptomatology: A Two-factor Model

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THE CLASSIFICATION OF THE SCHIZOPHRENIAS ACCORDING TO SYMPTOMATOLOGY: A TWO-FACTOR MODEL

by

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ABSTRACT

The purpose of this investigation was to examine a proposed two-factor model of schizophrenic symptomatology (Nicholson & Neufeld, in press). To achieve this aim, a semi-structured interview was administered to one hundred schizophrenic patients and a series of symptomatology scales were rated. Preliminary analysis indicated that these ratings were consistent with previous research in schizophrenia.

The item-level data from the scales was clustered to reduce redundancies in the large amount of data (95 schizophrenia variables) and 26 symptom clusters resulted. These symptom clusters were then rated and subsequently placed into four schizophrenia variable groups (paranoid, nonparanoid, positive, and negative). The four variable groups were put through a series of consistency tests to determine if their symptom clusters were related empirically as well as theoretically. The symptom clusters were then kept in a variable group if they met both types of relations.

Each of these variable groups were analyzed with Maximum Covariance Analysis to determine if they measured a single underlying construct. The resultant graphs were consistent with a dimensional model for the four groups. There was also some additional support for the dimensional model from comparisons of mathematically-independent categorical-model-based methods of calculating base rate estimates.

The divisions of symptoms into paranoid-nonparanoid as well as positive-negative offered strong support for the continuum of severity of disorder when they were combined. First, there was a large overlap between the two previously-hypothesized sets of divisions. Second, the

four variable groups all had Maximum Covariance Analysis results consistent with latent dimensional models. If the four groups were linked, then a single dimension of disorder would result.

These findings have significant implications for both future research and clinical practice. In particular, they offer strong support for a Bleulerian view of schizophrenia as a unified whole which has different presentations. As a result, research need not become primarily concerned with trying to explain differences between groups of schizophrenia patients but should look for commonalities amongst them. Also, the increasing recognition of the importance of dimensions in diagnosing mental disorders is discusssed.

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This work is dedicated with love to my wife, Cindy, and our children, Michelle and Timothy. Their support and sacrifices throughout this project has been unfailing. It is through them that I gained the necessary perspective that this project required and learned to identify life's true priorities.

When all is done, the help of good
counsel is that which
setteth business straight.

- Francis Bacon

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CHAPTER 1 - INTRODUCTION

The purpose of this investigation was to examine a proposed two-factor model of schizophrenic symptomatology (Nicholson & Neufeld, in press). Prior to any description of the model and how it was examined, it is important to understand the reasons for its development. First, it is necessary to describe some of the history of the classification of psychopathology and its current status, including a brief description of proposals for official systems that are to be formally introduced in 1993. General problems with these systems are also briefly described. To allow for a more thorough understanding of the problems of classification in psychopathology, the history of the diagnosis of schizophrenia is reviewed. This review involves early controversies in diagnosis as well as a description of the debate surrounding the use of symptom-based diagnosis for a disorder with a probable physiological basis. Six current diagnostic systems for schizophrenia are then described and compared with regard to their strengths and weaknesses. One result of these comparisons is the recognition of heterogeneity in the presentation of schizophrenia. Some of the methods of attempting to subtype the disorder according to these different presentations are then reviewed. One result of this review is the recognition that the paranoid/nonparanoid dichotomy has been the most consistently supported both clinically and in experimental research. Nevertheless, an examination of this division indicates that there are problems in this method of subtyping patients with schizophrenia.

In an attempt to deal with these limitations, a tentative reconceptualization was proposed for schizophrenia. This new model was

based on two independent factors: (1) Severity of Disorder and (2) Severity of Symptoms. This dimensional approach was shown to be consistent with a number of empirical findings regarding differences and associations between paranoid and nonparanoid symptomatology. The nature of dimensional formulations in psychopathology, for both symptoms and disorders, were also reviewed. The relative advantages of dimensional and categorical descriptions, as well as their combination, were then presented.

The present investigation is then introduced as a method of determining if the paranoid/nonparanoid distinction is best viewed as continuous or discrete. Two recently developed methods were employed to examine this question: maximum covariance analysis (Meehl & Golden, 1982) and the comparison of independent base rate estimates (Gangestad & Snyder, 1985). These methods have been employed in previous investigations to determine if dimensional models or categorical models are more appropriate to describe data sets. Their present investigation will involve investigations of whether sets of schizophrenic symptoms indicate the presence of a latent taxon in the data or if the data can be viewed as one single group.

A General Review of Modern Psychiatric Classification Systems

As noted above, it is important first to place the need for the proposed two-factor model in its proper historic context. Problems in the classification of psychopathology have been evident for centuries. Hippocrates, in the fifth century BC, generally is recognized as having the first recorded classification system for mental disorders (Alexander & Selesnick, 1966; Zilboorg, 1941). In it he included such disorders as epilepsy, mania (states of abonormal excitement), melancholia (states of

abnormal depression), and paranoia. He also recognized hysteria although he viewed it as a women's physical condition that resulted from a wandering uterus (Zilboorg, 1941).

Although there were some isolated attempts (e.g, Arataeus in the first century AD; Alexander & Selesnick, 1966), there was no general interest in the classification of disorders until the late seventeenth century (Singerman, 1981). During this time, for example, Francois Boissier de Sauvages published the 3-volume Nosolgie Methodique in which he outlined ten classes of disease. The eighth class was termed "Folie" and was divided into the four categories of psychopathology and each had a number of subdivisions. Zilboorg (1941) notes that, while they were orderly, there was no unity within each order (a characteristic that helps define modern classification systems). By the beginning of the nineteenth century, contemporary writers reported that almost every worker dealing with mental disease felt he had to offer his own classification system (Zilboorg, 1941).

In reviewing the history of official classifications, Spitzer and Williams (1985) identified the decennial census in the United States in 1840 as the first "official" classification system. It contained only one "mental illness" category which combined "Idiocy" and "Insanity". In the 1880 census, there were seven different categories (Mania, melancholia, monomania, paresis, dementia, dipsomania, and epilepsy). In 1889, Morel headed a commission for the International Congress of Mental Science which developed an 11 category classification system. Nonetheless, nineteenth and twentieth century psychiatrists in the United States generally were disinterested in (if not hostile towards) elaborate, systemized classification systems (Grob, 1991)

Emil Kraepelin is thought by many to have introduced the first modern classificatory system for psychopathology in the 6th edition of his Textbook of Psychiatry published in 1899. In this edition, he described 48 "forms of mental disease" under 13 major categories. These categories included the introduction of "manic-depressive insanity" and "dementia praecox". While not creating these categories, his texts were clear and outlined what the student should look for in the diagnosis of mental disease. His approach emphasized past and present symptoms of the patient and the patient's prognosis while it avoided theoretical arguments concerning etiology. For these resasons, this system of classification grew to prominence in the European psychiatric community.

In the years that preceded and followed the introduction of Kraepelin's system, classificatory schemes florished (Zilboorg, 1941). Unfortunately, different pockets of researchers and practitioners across both Europe and North America each developed their own terminology and nosology. The confusion soon became so great that conferences were held to develop uniformity in nomenclature and taxonomy (Grob, 1991). In 1917, the American Medico-Psychological Association (which later became the American Psychiatric Association) published the first edition of the Statistical Manual for the Use of Hospitals for Mental Disease. Its aim was to systematically collect data to serve as a basis for raising the standards of care for "the insane". Ten editions of this manual were published between 1918 and 1942.

In 1932, the New York Academy of Medicine developed their first edition of the Standard Classified Nomenclature of Disease. This nosology was later subsumed under the World Health Organization's (WHO's) International Classification of Diseases (ICD). The ICD

systems, as a whole, have been developed for the reporting of national and international statistics of morbidity and mortality for all known diseases. Its psychiatric section must, therefore, also follow this basic structure for statistical classification. As such, it aims to be an instrument of international communication, education, and research. With the advent of these systems, some of the confusion was lessened although a considerable amount still remained.

It is interesting to note that in Canada there was no classification scheme endorsed by the federal government until 1932. This "Dominion of Canada" system was used in the census and included six categories: Insane, mental defectives, epileptics, alcoholics, drug addicts, and all other types. The Insane category was subdivided into 23 subtypes, including Huntington's chorea, senility, "due to drugs", "psychoneurosis and neurosis", and "cerebral syphilus" (Dominion of Canada, 1932).

To further lessen the confusion of these different categories, the American Psychiatric Association began a major review of their system in the late 1940's and the early 1950's. Another more political reason for the review was theoretical (Grob, 1991). Before the Second World War, the majority of American psychiatrists worked in mental hospitals. As a result, most of the catagories reflected the views that mental disorders had a biological basis. During the war, a great number of psychiatrists were trained to deal with problems at "the front". By the end of the war, the American Psychiatric Association had more than doubled in membership. The emphasis in their training had been psychodynamic and psychoanalytic and the patient population they served was outpatient. These younger psychiatrists wanted their classification system to

reflect their patients and their theories on mental disorders. The result was the Diagnostic and Statistical Manual, Mental Disorders which appeared in 1952. This version has since been referred to as DSM-I.

The DSM-I divided mental disorders into two major groupings. The first represented cases where the symptoms resulted from (or were precepted by) an impairment of brain functioning (e.g., infection, drug, trauma, multiple sclerosis). The second group encompassed disorders resulting from an inability of the individual to "adjust" to the environment. This grouping was divided into two sub-categories: psychotic (e.g., manic-depressive) and psychoneurotic disorders (e.g., anxiety neuroses).

In 1955, the seventh edition of the ICD manual (ICD-7) was introduced but the mental disorders chapter was not updated. It continued to reflect the ICD-6 classification. This classification system was seen as inadequate by American psychiatrists because it did not include some disorders such as dementia and many of the personality disorders (Widiger, Frances, Pincus, Davis, & First, 1991). As a result, the WHO undertook the task of revising the ICD. An international review of classification systems was commissioned by the WHO and was led by Erwin Stengel. Its aim was to determine the reason for the lack of widespread international acceptance of the mental disorders section of the ICD 6. Stengel's (1959) review indicated that there were several very different classification schemes in use at the time. Many countries, including Canada, had their own official classification systems. Some countries would even have more than one system. For example, in the United States both the American Psychiatric Association and the War Department had their own system. In other

countries, there was no official system. In these instances, different theoretical orientations or different medical schools would teach different individual schemes. Stengel concluded that the lack of widespread acceptance of the ICD-7 was because many of the diagnostic terms were related to etiological, and therefore theoretical, schools of psychiatry. Stengel then made a series of suggestions for "requirements" for the ICD system to follow (e.g., the use of operational symptom-based definitions of disorders).

After many years and numerous conferences, the mental disorders chapter of the ICD-8 was published in 1968. The American Psychiatric Association was closely involved in its development. In that same year, the second version of the American Psychiatric Association manual was published (DSM-II). The DSM-II was closely based on the ICD-8 classification but the definitions were developed for use in the United States. These definitions were necessary because the WHO did not publish a glossary of definitions until 1972, 4 years after adopting the ICD-8. While several countries "officially" adopted the ICD-8, a review by Saugstad and Odergard (1983) indicated that few countires strictly adhered to the ICD-8 categories in practise. These reviewers proposed that the lack of clear instructions and definitions made its use difficult.

While many steps were taken to develop these two systems so they would be as comparable as possible, many differences remained. For example, psychotic and nonpsychotic organic brain disorders were viewed as two separate disorders in the ICD-8. Psychiatrists in the United States, however, did not make that distinction. Therefore, no distinction was outlined between the two disorders in the DSM-II. Such

differences between the systems led to continued confusion and often to an inability to generalize between European and North American research.

The nosological systems have continued to develop since that time. In 1975, the ICD-9 (with accompanying glossary) was published and went into effect in 1978. There were, however, few changes from the ICD-8. Unlike its predecessors, the ICD-9 was followed by a systematic assessment of reliability and validity of its categories. Unfortunately, the majority of studies concluded that, when coded by mental health professionals, several of the categories were unreliable (e.g., Sytema, Giel, Ten Horn, Balestrieri, & Davies, 1989; Torgersen, Rosseland, & Malt, 1990).

In 1980, the American Psychiatric Association released the third edition of its manual (DSM-III), which differed radically from the previous versions. Representative advisory committees were formed for each of 12 major areas of classification, for the multiaxial system, and for the technical glossary of terms. Each of these areas drafted the text for the diagnostic categories in their area of expertise. While the DSM-III aimed at being as compatable as possible with the ICD-9, this was not the primary goal (Spitzer & Cantwell, 1980). In reviewing the DSM-III, Klerman (1983) reported that it represented or contained five innovations. First, it reintroduced the notion that there could be multiple, separate disorders. Second, there were operational definitions for disorders. Third, the criteria were based (for the most part) on manifest descriptive psychopathology rather than inferences or criteria from presumed causation or etiology. Fourth, the DSM-III had field trials to assess reliability prior to publication. Fifth, a multiaxial system was introduced which assisted in separating

developmental and personality disorders (Axis II), physical disorders (Axis III), social stressors (Axis IV), and the level of general adaptive functioning (Axis V) from the underlying syndromal diagnosis (Axis I).

Controversies exist in almost every area of the DSM-III. A number of criticisms are general or philosophical (e.g., Kirk & Kutchins, 1992; Salzinger, 1986; Wortis, 1982). Some criticism, leveled since its development, has been aimed at procedural issues surrounding the manual (Millon, 1987; Taylor, 1983). Other criticism stems from social issues such as gender equality in that the system has been accused of reflecting primarily a male perspective on disorder (Adler, Drake, & Teague, 1990; Kaplan, 1983). Furthermore, the composition and operationalization of each axis has been called into question. For example, is the Axis II section of childhood and adolescent psychopathology based firmly enough on empirical research and, if not, does it contain numerous obsolete and ill-conceived categories (Bemporad & Schwab, 1986; Quay, 1986)? Are the Axis II personality disorders sufficiently discriminable (Bell & Jackson, 1992)? Does Axis III present an overly simplistic view of the relation between physical disorders and psychiatric disorders (Leigh, Price, Ciarcia, & Mirassou, 1982)? Does the importance of Axis IV, the level of psychosocial stressors over the past year, compensate for the unreliability of its assessment (Rey, Stewart, Plapp, Bashir, & Richards, 1988). Does the inclusion of Axis V's measurement of "Adaptive Functioning" relate more to social conformism than to medicine (Hoenig, 1981)? Such challenges have been varied in both their nature and their sources, and there is

little consistency. That is, no single set of general criticisms has had near universal voicing by the DSM-III's critics.

Research has also shown that the DSM-III has had a major impact. Strauss, Yager, and Strauss (1984) surveyed 138 "experts" in psychiatry and asked them to name the most important publications of the years 1970 to 1980. The DSM-III was listed twice as often as the second most-listed text (Comprehensive Textbook of Psychiatry). At an international level, there was some initial reluctance to embrace the DSM-III completely but the majority of opinion was in its favour (Spitzer, Williams, & Skodol, 1983) including that from Canada (Engels, Ghadrian, & Dongier, 1983). Part of this international acceptance is due to the efforts by the originators of the DSM-III to maintain some resemblance to the ICD-9 in the coding while allowing for several differences in diagnostic criteria (Skodol & Spitzer, 1983). Thompson, Green, & Savitts (1983) developed a "crosswalk" to allow for translations between the two systems which then allows a clinician to diagnose according to the DSM-III but report with an ICD-9 code. More recent research indicates that the international acceptance of the DSM-III system has increased (Helzer & Canino, 1989; Maser, Kaebler, & Weise, 1991).

In 1987, the DSM-III-R was released. It was intended as an interim solution during the development of the DSM-IV (Spitzer & Williams, 1987). This new manual includes several minor changes to the nosology of the DSM-III as well as a few major changes (e.g., dropping "Ego-Dystonic Homosexuality"). It also allows for more co-morbidity of disorders, and includes a new section of four "proposed diagnostic categories needing further study". Some researchers have hailed

the DSM-III-R as making a good system even better (e.g., Ben-Tovim, 1988; Kendell, 1988; Rutter, 1988).

Several researchers, however, have questioned the clinical importance of the changes made in arriving at the DSM-III-R (e.g., Cooper & Michels, 1988; Frances, Widiger, & Pincus, 1989; Swartz, 1989; Zimmerman, 1988). Some critics have charged that many of the proposed diagnostic categories (e.g., self-defeating personality disorder, late luteal phase dysphoric disorder) continue to reflect the significant gender bias referred to above (Holden, 1986). Others believe that the DSM-III-R introduces too many changes with too little evidence and that the result is unnecessary confusion and frustration (Rey, 1988). Nonetheless, the adoption of the DSM-III-R as an official diagnostic system has been widespread (Thompson & Pincus, 1989).

In Europe during this time, the "Mental, Behavioural, and Developmental Disorders" chapter (i.e., Chapter V (F)) of the prosposed ICD-10 was being developed under the supervision of Norman Sartorius (1988), Director of the WHO Division of Mental Health. This classification system was published in 1992 and, following reorientation courses, a formal introduction is scheduled for 1993 (Bramer, 1988). In comparison to the ICD-9, there are numerous important changes (Cooper, 1988, 1989). First, it is much larger than the system it replaces. The first character in its coding system was previously a single digit but is now a letter. This change allows for a considerable increase in the number of diagnostic categories. Second, the differentiation between psychoses and neuroses is no longer the fundamental organizing principle. Third, disorders with common properties are now grouped together. For example, "Schizotypal Disorder" is now in the same group

as schizophrenia and delusional disorders. Fourth, different versions of the manual will be produced for different purposes. Although the categories are exactly the same, the amount of detail and manner of presentation are tailored differently for different purposes. Fifth, the ICD-10 follows the lead of the DSM-III and includes detailed diagnostic criteria and points of differential diagnosis. Finally, since the ICD-10 was prepared in coordination with the future release of the DSM-IV, it is hoped that their diagnoses will be as compatable as possible (Cooper, 1988). Initial research on the interrater reliability of the ICD-10 generally indicates that it is seen as better than its predecessars (Ellis, Welch, Purdie, & Mellslop, 1990) and not significantly different from the DSM-III-R (Mellslop et al., 1991). Nonetheless, it is evident by both the continuing changes and the plans for future changes, in both the APA and the WHO systems, that problems have continued to plague the classification of psychopathology (Frances et al., 1989, Frances, Widiger, et al., 1991; Klerman, 1986).

Shortly after the DSM-III-R's publication, the development of the DSM-IV was announced (Sabshin, 1988). The primary reason for its development is to meet the requirements of an international treaty to have the official United States diagnostic systems compatable with the ICD (Frances, First, Pincus, Widiger, & Davis, 1990). The Task Force on DSM-IV has described how the development of criteria will be obtained and reviewed via three interactive phases (Widiger, Frances, Pincus, Davis, & First, 1991). In the first stage, 175 comprehensive and systematic literature reviews will be conducted to provide a framework for the working groups to address specific diagnostic issues (Widiger, Frances, Pincus, & Davis, 1990) as well as issues surrounding the

several axes (Goldman, Skodol, & Love, 1992; Williams, Goldman, Gruenberg, Mezzich, & Skodol, 1990). The second stage involves the over 40 re-analyses of data from multiple relevant existing but unpublished data sets. This research usually required the collaboration of several investigators at different sites. This stage allows for the analysis of issues not adequately covered by the literature searches (e.g., Late Luteal Phase Dysphoric Disorder; Hurt et al., 1992). The third stage involves field trials to assess the proposed DSM-IV categories, DSM-III, DSM-III-R, and ICD-10 through the use of surveys, videotape reliability studies, and focused field trials (Widiger et al., 1991). Each field trial will involve five to ten different sites with approximately 100 subjects at each site (Task Force on DSM-IV, 1991). Publication is planned for the results from all three of these stages. Guidelines have also been proposed for the DSM-IV Working Groups to follow for diagnostic inclusion and exclusion (Blashfield, Sprock, & Fuller, 1990; Pincus, Frances, Davis, First, & Widiger, 1992).

Considerable effort has been made to make this process as open as possible (Francis, Widiger et al., 1991). Special issues of journals have been devoted to the DSM-IV (Andreasen, 1991; Barlow, 1991; Schwartz, 1991). Also, Hospital and Community Psychiatry has a column appearing approximately once every 2 months in which a DSM-IV Working Group discusses the issues and its results to date (Francis, First, Pincus, Widiger, & Davis, 1990). In 1991, the Task Force on the DSM-IV published the DSM-IV options book: Work in progress to "help ensure that (they) are receiving the widest possible input of data and opinion and that (they) are not missing inconsistencies, errors, or potentials for misuse" (p. A:1). By such openness, the DSM-IV developers hope to

avoid the perception, present with the developments of both the DSM-III and DSM-III-R, that the document was the result of closed-door secret deliberations.

Criticism has already developed for this proposed scheme. Much of this criticism centres on the view that not enough time has elapsed since the last set of official changes (Kaplan & Sadock, 1989; Zimmerman, 1988, 1990). As a result, there has been insufficient time for data to accumulate on the last set of diagnoses (Blashfield, Blum, & Pfohl, 1992). There is also the belief that the development of a third major American system in 13 years would lead to confusion as clinicians tend to employ the system learned in their training (Ellis & Mellsop, 1990; Zimmerman, Jampala, Sieber, & Taylor, 1991). Finally, there are critics who argue that the process is flawed at some level, either theoretically (Carson, 1991) or politically (Caplan, 1991). These criticisms are often the same that these individuals have made about the DSM-III or DSM-III-R (e.g., Carson, 1990). Thus, the developers of the DSM-IV have not been able to address all of the concerns about the earlier versions but have attempted to deal with much of the earlier criticism.

Not only have there been problems with these various systems, but problems also exist at the level of the major established categories within these systems. For example, this continuing development and confusion is evident in "dementia praecox", now more commonly referred to as schizophrenia. The assessment of schizophrenia has been an area of considerable debate, discussion, and disagreement since Kraepelin first outlined it. Such difficulties not only include its nosological definition but also in subtype categorization. Many of these

difficulties are the result of different definitions and assessment of the relevant symptoms. In the following sections, the history, strengths, and weaknesses of schizophrenic diagnosis, and subtype categorization will be outlined.

The Diagnosis of Schizophrenia

While Kraepelin developed the diagnostic system, which included the first definition of schizophrenia as we know it today, the schizophrenic syndrome has been known for thousands of years. Sanskrit writings from the 14th century B.C. have references to the disorder (Doran, Breier, & Roy, 1986). Some contemporary medical historians have reviewed the lives of historical figures (e.g., Socrates) and attempted to find evidence for schizophrenia in their lives (e.g., Zilboorg, 1941). German and French psychiatric texts in the 19th century include several references to variants of schizophrenia (Wilkinson, 1987). Laseaque used the term delire de persecution to describe a patient in 1852. In that same year, Morel described a "demence precoce" which was marked by severe intellectual deterioration, withdrawal, and bizarre mannerisms that started in adolescence. In 1871, an illness which resulted in a "silly" deterioration starting in puberty was described by Hecker. In 1874, Kahlbaum decribed an equivalent to catatonic schizophrenia which was termed "tension insanity" (Cancro, 1985).

In 1893, Kraepelin used the term "dementia praecox" to describe a narrow group of hebephrenic psychotic disorders. In November 1898, he presented a modified dementia praecox which was expanded from its previous version. It stood apart from previous descriptions by its grouping together of three forms of dementia praecox: catatonic, hebephrenic, and paranoid. Common to these forms were several symptoms,

including progressive mental deterioration (i.e., dementia) and onset during puberty or adolescence (i.e., praecox). These common symptoms allowed the forms to be organized under the one major category. This description was then expanded and placed in the 1899 edition of Kraepelin's Textbook of Psychiatry.

He emphasized in that description that prognosis was an important variable and related to symptomatology and course (WHO, 1973). These changes were so radical that his views were not initially adopted. Some opponents disagreed with his view that over 80% of patients had a severe intellectual deterioration (Zilboorg, 1941). Others were resistant to the difficult necessity of observing the course of a patient before making a diagnosis or making a prediction (Grebb & Cancro, 1989).

Kraepelin revised his categories several times in the years before his death in 1927, the last complete version appearing in 1913. In this version he described dementia praecox as "a series of states, the common characteristic of which is a peculiar destruction of the internal connections of the psychic personality. The effects of this injury predominate in the emotional and volitional spheres of mental life" (p. 3, 1913/1919,; italics added). Thus, Kraepelin never hypothesized that there was only one single clinical entity which was responsible for all of these groups. Instead, he grouped together a series of poorly understood psychotic syndromes. This use of a common overarching diagnostic category allowed for general predictions as to the cause and outcome of the three syndromes. It also allowed for variations which did not fit exactly into his schema.

During the time in which Kraepelin was revising his texts, Eugen Bleuler's (1911/1950) text on schizophrenia appeared. It has made a

major impact ever since its publication for several important reasons. First, he widely introduced the term "schizophrenia" to overcome the difficulties inherent in the use of the term "dementia praecox" (although he is reported to have employed the term "schizophrenia" as early as 1908 in journal articles; Berrios, 1987; Sass, 1987). Employing this term also allowed Bleuler to emphasize the disintegration of personality due to a loosening of associations which he viewed as the most important characteristic of the disorder. In 1912, the term "schizophrenia" was first employed in an English-language medical journal, The Lancet, in an anonymous review of one of Bleuler's texts (Simpson & Weiner, 1989) and has been used increasingly since that time. Second, he reinforced the three categories which were put forth by Kraepelin and added a fourth: "simple" schizophrenia. Third, he posited that the groups of schizophrenias should be viewed as a single clinical entity as they had four primary characteristics which were common to them all (disorders of association, autism, ambivalence, and affect). It has been Bleuler's view which has since dominated the majority of theory and research on schizophrenia from the publication of the English translation of his text in 1950 until the development of the DSM-III in 1980 (Andreasen, 1989a; Cromwell, 1975).

Symptom-based classification systems. At this point, it should be noted that these previous groupings were all based on symptomatology. The use of this method at the time can be explained by the fact that the interests of these early researchers were almost solely in behaviour and symptomatology. With the recent advances in biochemistry and imaging procedures, there has been considerable hope for the acceptance of such biological techniques for the diagnosis of psychopathology, with special

emphasis placed on schizophrenia (e.g., Buschbaum & Haier, 1987; Mesulam, 1990). These techniques include newer imaging techniques such as magnetic resonance imaging (MRI; e.g., DeMyer et al., 1988), positron emission tomography (PET; e.g., Andreasen et al., 1988), evoked potential (EP) mapping (e.g., Faux et al., 1988) and mapping from electroencephalagraphic data (EEG; e.g., Karson, Coppola, Daniel, & Weinberger, 1988). Biochemical research has focussed on monoamines, such as dopamine (DA; Carlton & Manowitz, 1984), and serotonin (5HT; Jackman, Luchins, & Meltzer, 1983), but have also studied a variety of other compounds such as platelet monoamine oxidase (MAO), neuropeptides, lymphocytes, norepinephrine, and endogenous opiates. The research to date repeatedly indicates some form of structural or neurochemical abnormality (Meltzer, 1987). Because of the advent of these techniques, some authors assert that symptomatology-based diagnoses are out-dated in the assessment of schizophrenia (e.g., Zubin, 1986).

Nevertheless, while such advanced techniques show promise, they have not yet lived up to their expectations (Andreasen, 1988; Szymanski, Kane, & Lieberman, 1991). Reviews have indicated that "a clear association with biochemical abnormalties and schizophrenia has not yet been found" (p. 514, Karson, Kleinman, & Wyatt, 1986) and "none of the brain imaging techniques have found incontrovertible evidence of an anatomical or functional defect" (pp. 677-678; Young & Williamson, 1986). Thus, while there are some interesting findings and promising new areas of research, the future of biological research into schizophrenia remains guarded. As a result, it has been proposed that greater emphasis be placed on phenomenology in schizophrenia research

(Mortimer, 1992). Therefore, the assessment techniques and nosological networks which are reviewed will be restricted to symptomatology.

One further point should be made with regard to the use of such classification systems. Even though there is very strong evidence for a physiological basis for schizophrenia (Berman & Weinberger, 1987), that should not rule out its definition and classification by symptomatology (Carpenter, Strauss, & Bartko, 1974; Corning & Steffy, 1979). In reviewing taxonomizing in psychopathology and medicine, Meehl and Golden (1982) made the point that "the entity is not the syndrome" (p. 134). In other words, the individual symptoms in and of themselves do not constitute the disorder. Instead, they are only imperfect indicators of an underlying psychopathological entity. Yet, even though the relation is imperfect, it has not stopped medical diagnosis. The diagnosis of medical disorders is done primarily by the investigation of symptoms. For example, in the early part of this century, polio was not diagnosed by the measurement of the poli virus in an individual. It was diagnosed by the symptoms which were present. As such, it gave evidence for the diagnosis of an underlying disorder by the imperfect measurement of observable symptoms, not by measurement of "the disorder itself".

The definition of schizophrenia by symptomatology is consistent with a structural or neurochemical basis for the disorder. Such symptom-based classification continues for other physiologically-based disorders which result in behavioural abnormalities. This method of taxonomy is especially evident in neuropsychology. For example, "aphasia" is a "neurologically central disturbance of language characterized by paraphasias, word finding difficulty, and variably impaired comprehension (p. 2; Kertesz, 1979). Thus, aphasia, while it

is the result of a central nervous system disorder (e.g., stroke, episode of epilepsy), is diagnosed by the behaviours which result from it. Tests for aphasia, such as the Western Aphasia Battery (WAB; Kertesz, 1982) or the Boston Diagnostic Aphasia Examination (BDAE; Goodglass & Kaplan, 1984), use symptoms, such as decreased verbal fluency or impaired repetition, for the diagnosis of aphasia.

These same symptoms are then used to classify patients into an aphasic subtype. For example, the WAB would classify someone with impaired verbal fluency, naming ability, and repetition ability but with intact verbal comprehension as a "Broca's aphasic". As such, the symptoms diagnose the underlying entity, though it is recognized that these symptoms are not the entity in and of itself. In other words, it is recognized "that the disease entity does not consist of nothing but the symptoms" (p. 135; Meehl & Golden, 1982; italics in original).

Many modern classification systems in neuropsychology were developed by using a variety of statistical techniques, such as cluster analysis (Kertesz & Phipps, 1977) or discriminant function analysis (Goodglass & Kaplan, 1984). The resultant entities were then compared to those groups which have been previously discussed in the literature. For example, Paul Broca's identification in the 19th century of a nonfluent aphasia parallels that of the type outlined above as Broca's aphasia. This grouping continues to be symptomatology-based, even though it has long been recognized that it results from damage to the posterior portion of the frontal lobe of the patient's dominant hemisphere. In fact, this portion of the hemisphere is so strongly related to this specific language disorder that it is often referred to as Broca's area.

Such localization, however, is not always possible. Several neuropsychological dysfunctions which result from cortical or subcortical damage have not been localized although they represent a single syndromal entity (e.g., hemispatial neglect; Heilman, Watson, Valenstein, & Damasio, 1983). Likewise, the subtyping of syndromes has not always led to more accurate localization of corresponding neurological damage (e.g., subtypes of Wernicke's aphasia; Kertesz, 1983b; Kirshner, Casey, Henson, & Heinrich, 1989). Even though specific physiological mechanisms may or may not be responsible for the deficits noted in the majority of neuropsychological syndromes (Kertesz, 1983a), the behavioural "symptomatology" deficits continue to govern classification. Therefore, to do the same in psychopathology for the identification, classification, and investigation of disorders such as schizophrenia would not be unique.

On balance, there is considerable precedent for focusing taxonomic perspectives on behavioural "symptomatology" data. Legitimacy of such focus has not depended on the ready localization of physiological substrates. On the other hand, the claim of "constituting the disorder" is not arrogated to a set of behaviours; nor are taxonomies comprising constellations of behaviour in any way incompatable with identifiable biological sources.

Diagnostic Systems for Schizophrenia

Six diagnostic systems which centre around or include schizophrenia will be reviewed in this next section. While it is recognized that several other diagnostic systems exist (e.g., Langfeldt, 1960), these six have been widely employed and continue to have a wide impact. These diagnostic systems are the First-Rank Symptoms, the St. Louis (Feighner)

criteria, New Haven Schizophrenia Index, 12-Point Flexible System, Research Diagnostic Criteria, and DSM-III/DSM-III-R. Included in these reviews will be the results of some of the research which has directly compared some of these systems in their diagnoses of the same samples of patients.

First-Rank Symptoms. From his clinical experience, Kurt Schneider (1959) developed a list of basic symptoms which he viewed as being frequent enough in schizophrenia to be useful as diagnostic criteria (see Appendix A). If one of these First-Rank Symptoms (FRS) were present, and there was no organic disorder present which could be responsible, than the clinician could diagnose schizophrenia over cyclothymia. The listing of these specific symptoms occured because they were easy to ascertain and they were important in diagnosis. The FRS did not have any additional etiological or theoretical value according to Schneider. Early research was quite supportive of the use of this symptom checklist (Mellor, 1970).

Subsequent research, however, has failed to support the FRS as a diagnostic system for schizophrenia. For example, research has not supported the view that these symptoms are more effective than non-FRS psychotic symptoms, such as visual hallucinations, in the identification of schizophrenics (e.g., Silverstein & Harrow, 1981). Furthermore, some research indicates that up to 25% of patients with primarily affective disorders may be diagnosed as schizophrenic given these criteria (Carpenter, Strauss, & Mulch, 1973). Finally, neither the presence, absence, or number of the FRS has been shown to relate to outcome (e.g., Bland & Orn, 1980). Taken together, the research on the FRS has

only given it limited support for its use in the identification of schizophrenia (Hoenig, 1984; Overall, 1981).

St. Louis (Feighner) Criteria. In 1972, a group of researchers in St. Louis, namely Feighner, Robins, Guze, Woodruff, Winokur, and Munoz, outlined diagnostic criteria for 14 psychiatric illnesses (see Appendix B). Their judgements were based on the clinical research found in the literature and in their own experience. This set of criteria has since become known as either the Feighner criteria or the St. Louis criteria. Its major influences were in the recognizing a limit to age of onset (i.e., before age 40) and specifying a minimal duration of illness (i.e., six months). Its influence was considerable during the 1970's and early 1980's.

Blashfield (1984) described the St. Louis criteria's most "remarkable feature" as its impact on research in the years that followed. By means of a citation analysis, Blashfield determined that it received over 1650 citations in the ten years that followed, with over 200 citations in 1982 alone. In contrast to this level, the average citation count for articles published in 1973 was only 2.1 by 1976. While the use of the St. Louis criteria has declined somewhat in recent years due to the increased use of the Research Diagnostic Criteria and the DSM-III, it remains popular (125 citations in 1990; 113 citations in 1991).

Blashfield (1982) argued that many of these citations, however, were due, at least in part, to a variety of external, extrascientific effects. Such effects include the high number of researchers produced by that particular St. Louis research centre as well as the centre's high number of self-citations. Other researchers have pointed out,

however, that the St. Louis criteria also filled a very strong need which developed shortly before its publication (Katz, 1982; Kendell, 1982; Strauss, 1982).

In comparisons to other systems, the utility of the St. Louis criteria continues to be supported. For example, the criteria have higher reliability than most other systems (Helmes, Landmark, & Kazarian, 1983). Furthermore, they have been shown to identify a more narrow group of patients as schizophrenic than many other systems (e.g., Strauss & Gift, 1977). Follow-up studies have indicated that these schizophrenics would more likely retain their symptoms, social handicaps, and diagnosis than those schizophrenics diagnosed by other systems (e.g., Helzer, Brockington, & Kendell, 1981; McGlashan, 1984). Thus, the criteria demonstrate a low false-positive rate. (It is recognized that the criteria may also demonstrate a high false negative rate. This bandwidth-fidelity argument is discussed below.)

In reviewing the numerous studies done on the reliability, validity, and specificity of the St. Louis criteria, Fenton, Mosher, and Matthews (1981) concluded that it was successful in defining a homogeneous group of schizophrenic patients. Nevertheless, recent research on the St. Louis criteria has noted the lack of internal consistency in its rules for diagnosis (Winokur, Zimmerman, & Cadoret, 1988). It seems that at present, the greatest legacy of the St. Louis criteria is in how other systems (e.g., DSM-III-R) have included portions of it.

New Haven Schizophrenia Index. Astrachan et al. (1972) reported on the development of a symptom checklist to discriminate schizophrenics from nonschizophrenics in treatment settings. The team of clinicians reviewed the literature to determine which symptoms had been identified

consistently as being associated with schizophrenia by both clinicians and researchers. The symptoms had to be easily retrieved from patient files and reliably rated. This checklist, termed the New Haven Schizophrenia Index (NHSI; see Appendix C), was tested to determine its accuracy in diagnosing schizophrenics in comparison to a second patient group composed of diagnoses which might be confused with schizophrenia (e.g., organic brain syndrome, delusional depression). The NHSI had a true-positive rate of 87.4% and a false-positive rate of 13%. The scale was then reviewed and certain items dropped which did not differentiate groups. On a cross-validation study with similar samples, the true-positive rate was 89.7% and the false-positive rate was 14.4%. According to Astrachan et al., the NHSI was shown to be reliable, valid, easily scored, and adaptable to computer analysis.

The NHSI has not been employed often since that time. Even though the inventory has been shown to have very high levels of reliability (e.g., Endicott et al., 1982), it repeatedly has been demonstrated that it classifies the greatest number of patients as schizophrenic (e.g., Gift, Strauss, Ritzler, Kokes, & Harder, 1980; Stephens, Astrup, Carpenter, Shaffer, & Goldberg, 1982). Furthermore, this wide range of schizophrenic patients has led to the NHSI repeatedly demonstrating low predictive validity when compared to other systems. For example, Astrachan, Brauer, Harrow, and Schwartz (1974) re-applied the checklist to 132 patients from their original schizophrenic patient group. Their results indicated that the patients who were diagnosed as schizophrenic by the NHSI were quite heterogeneous in their outcomes. Further research has suggested that this hetereogeneity is the result of schizoaffective and manic patients being classified as schizophrenic

(McGlashan, 1984). Because of these problems in validity, the LSI has rarely been employed as a diagnostic tool.

12-Point Flexible System. In 1973, Carpenter, Strauss, and Bartko reported on the results of the International Pilot Study of Schizophrenia (IPSS). The IPSS, which had been sponsored by the WHO (1973), was a nine country, 1121 patient investigation for diagnostic symptoms of schizophrenia. These countries, spread across five continents, were included in the study so as to bring a variety of different criteria for schizophrenia together in a search for common symptoms. All patients were given the same psychiatric examination, the Present State Examination (PSE; Wing, Birley, Cooper, Graham, & Isaacs, 1967), and then they were classified according to the ICD-8. A discriminant function analysis was then done on half of the sample to indicate which symptoms best discriminated schizophrenics and nonschizophrenics. A validation of the results was then conducted on the second half of the sample. Results indicated that twelve of the symptoms best discriminated the two groups (see Appendix D). These twelve symptoms were then collected as the 12-Point Flexible System.

This system was termed "flexible" because it allowed different numbers of symptoms to be employed in judging the presence of schizophrenia. As Table 1 indicates, if more symptoms are chosen, the true-positive rate decreases. Corresponding to this decrease, however, is a concomitant greater decrease in the false-positive rate. (These estimates of identification rates were based on the results of classifications which were cross-validated across two halves of the original sample.) Carpenter, Strauss, and Bartko concluded that the international nature of the study, the statistical cross-validation, and

Table 1

Percentages of Patients Diagnosed as Schizophrenic by the 12-Point Flexible System

Number of Symptoms	Schizophrenic		Nonschizophrenic	
	Cohort A	Cohort B	Cohort A	Cohort B
4 or more	91%	91%	28%	38%
5 or more	80%	81%	13%	22%
6 or more	66%	63%	4%	6%
7 or more	44%	39%	1%	1%
8 or more	23%	20%	0%	0%

its flexibility would result in the 12-Point Flexible System becoming a useful diagnostic instrument in future research.

Carpenter, Bartko, and Strauss (1980) later reviewed the research which had been conducted using the 12-Point Flexible System and placed several limitations on the utility of the system. First, their system was prepared using acute schizophrenics and is less suited for distinguishing chronic schizophrenia from nonpsychotic disorders. Second, to lower the amount of false-positives, the Flexible System should not be used by itself, but only in conjunction with a full clinical diagnosis. Third, the 12-Point Flexible System was a poor predictor of outcome. Both premorbid functioning and established chronicity were better predictors of future course and oucome. Finally, Carpenter, Bartko, and Strauss warned that manic patients are especially difficult to separate from schizophrenics when using the Flexible System. There was considerable enthusiasm for the 12-Point Flexible System when it first appeared. Yet, even though this enthusiasm subsequently led to considerable research, this research has failed to support the widespread use of the systeem.

Research Diagnostic Criteria. Spitzer, Endicott, and Robins (1978) developed a refinement and extension of the St. Louis criteria to be used for research purposes. The Research Diagnostic Criteria (RDC) describe 25 major diagnostic categories and numerous subtypes within these categories (e.g., 11 subtypes of major depressive disorder). The authors of the RDC gave special attention to the reliability of their diagnostic system. They attempted to define their criteria clearly, even leaving out traditional criteria which could not be objectively and reliably defined (e.g., ambivalence). Furthermore, Endicott and Spitzer

(1978, 1979) developed a structured interview, the Schedule for Affective Disorders (SADS), to reduce variance and increase reliability when diagnosing a psychiatric patient. With these changes, Spitzer and his colleagues hoped the RDC would eliminate many of the psychometric problems previously inherent in psychiatric research.

The definition of schizophrenia which is outlined by the RDC contains the most extensive criteria written up to that time (see Appendix E). The definition relies heavily upon those symptoms outlined previously by the FRS and the St. Louis criteria. The reliability of the RDC's diagnosis of schizophrenia has varied across studies, but generally, it has been higher than other diagnostic systems (Fenton et al., 1981). The rigorousness of the definition also results in streamlining the means of which patients can be termed schizophrenic, with the result that a smaller range of patients are identified (e.g., Gift, Strauss, Ritzler, Kokes, & Harder, 1980). Furthermore, research indicates that the RDC's predictive validity is equal to, or better than, that of other diagnostic tools and systems (Doran et al., 1986). Thus, the RDC was probably the best system available at that time for the diagnosis of schizophrenia.

DSM-III/DSM-III-R. The DSM-II diagnosis of schizophrenia (see Appendix F) was based largely on Bleuler's theories of schizophrenia (Andreasen, 1989a). Schizophrenia researchers, however, often had difficulties with this diagnosis. For example, Blashfield (1973) had a group of 55 clinicians attempt to diagnose a series of artificial "patients" according to the DSM-II subtype criteria. He found that many subtypes were redundant in their usage by these clinicians (e.g., latent and residual schizophrenia). He also found that the interrater

reliability for these diagnoses were "quite poor". Blashfield concluded that the results suggested that the system was not reliable and could not provide adequate coverage of true schizophrenic patients. As a result of such difficulties, researchers would sometimes develop their own diagnostic criteria (e.g., NIMSI).

During the 1970's, American psychiatry was affected by three important developments that would greatly influence the DSM-III in its development (Andreasen & Flaum, 1991). First, international studies repeatedly indicated that American definitions of schizophrenia were much broader than the definitions found elsewhere (e.g., Kuriansky, Deming, & Gurland, 1974; WHO, 1973). Second, research had begun placing an increasing emphasis on diagnostic reliablity (e.g., Spitzer & Fleiss, 1974). Third, there was an increasing emphasis on including the course of disorder with the cross-sectional information in making a diagnosis (e.g., Feighner et al., 1972).

Robert Spitzer was put in charge of the development of the DSM-III by being made Chairman of the American Psychiatric Association's new Task Force on Nomenclature and Statistics in 1974. It was decided by the Task Force that the new DSM-III would be a modification of Spitzer's RDC because the specific diagnostic criteria of the RDC repeatedly had been shown to be equal to, or superior to, other systems available at that time for general nosology (Spitzer, Andreasen, & Endicott, 1978). Considerable research, however, had been undertaken since the initial development of the RDC (Spitzer & Williams, 1985). In addition, the Task Force would be forced to take into account the clinicians' requirements of practice and not just the needs of researchers (Spitzer, Endicott, & Robins, 1975). The enormity of the

task resulted in numerous advisory committees being formed (e.g., Impulse Control Disorders, Eating Disorders) and a series of draft versions and field trials were undertaken. The time taken for the development of the DSM-III increased 50%, from the projected four years to six years.

Skodol and Spitzer (1983) described the aims and methods of the DSM-III development of the diagnosis of schizophrenia (see Appendix G). The major aim was to narrow the definition of schizophrenia. This change was made by eliminating ambiguous subtypes (e.g., latent schizophrenia) and classifying such individuals elsewhere (e.g., schizoid personality disorder). Also, the criterion was added that a minimum six month duration of symptoms must be present. This criterion was added to limit the number of brief psychotic reactions misdiagnosed as schizophrenia. These new restrictions allowed for the identification of a more homogeneous patient population with a tendency for onset in early adult life, recurrent episodes, increased prevalence among family members, severe functional impairment, and differential response to somatic therapies (Spitzer, Williams, & Skodal, 1980).

As outlined earlier, when the DSM-III was finally published in 1980 there was considerable controversy. Early reports, however, indicated high test-retest and joint interviewer reliability for most categories, including schizophrenia (Kappa = 0.82 for both; Spitzer, Forman, & Nee, 1979). These reliabilities were considerably higher than those reliabilities reported for the previous editions of the DSM.

Later research offered further strong validation for the DSM-III in a variety of areas (Kendler, 1987). For example, Stephens et al. (1982) reported that employing the DSM-III resulted in a narrow definition of

schizophrenia. When it was compared to eight other diagnostic systems, however, the DSM-III had the highest correlation with a variety of other follow-up measures. Thus, the DSM-III definition of schizophrenia resulted in a narrower band of patients with a greater probability of poor outcome (Moller et al., 1989). Because of its reliability and predictive ability, the DSM-III was listed as the favored diagnostic system in numerous studies which have compared available systems (e.g., Landmark, Cernovsky, Merskey, & Leslie, 1986; McGlashan, 1984).

This support should be tempered by the realization that there is criticism of the DSM-III definition of schizophrenia, especially in its relation to other diagnostic systems such as the DSM-II (Goodman, 1989) and the ICD-8 (Wittchen, Semler, & von Zerssen, 1985). For example, Coryell and Zimmerman (1987) surveyed the changes from the St. Louis criteria to the RDC to the DSM-III. In comparing the systems, Coryell and Zimmerman concluded "that the rate of revisions had outpaced the rate at which investigators in the field had generated and replicated the data on which such changes should be based" (p. 1473). They suspected that many of these changes were often based on a single study. Also, the requirements of six months duration was viewed by many as being arbitrary and only resulting in the "obvious fact" that "chronicity predicts chronicity" (p. 348; Spitzer & Williams, 1983). While there was considerable difficulty in developing this rule (cf. Spitzer, Andreasen, Endicott, & Woodruff, 1978), recent research indicates that this criterion alone does not predict poor outcome (Helzer, Kendell, & Brockington, 1983). There were also questions raised concerning other aspects of the criteria such as the upper age limit of 45 years for the initial development of schizophrenia (Spitzer

& Williams, 1983). There have also been concerns regarding findings that suggest that certain social groups (e.g., blacks) are more likely to be classified as schizophrenic (Pavkov, Lewis, & Lyons, 1989). Finally, another form of criticism is that the present definition is too restrictive because it does not allow signs that are rare or that are difficult to quantify reliably (e.g., anhedonia; Meehl, 1986). Thus, while it is the most favored diagnostic system at present, it is not without its critics (cf. Millon & Klerman, 1986).

The diagnostic criteria for schizophrenia in the DSM-III differ little from the criteria in the more recent DSM-III-R. First, the symptoms in section A must now be present for at least one week (unless they are treated successfully). Second, the criteria in section B have been revised to take into account possible childhood onset. Third, the DSM-III-R now avoids the term "deterioration". Fourth, the need for onset before age 45 has been eliminated as its validity has not been supported by recent research. Fifth, the diagnostic criteria now include a recognition of a "stable" type, in which the criteria for A and B have been present throughout all phases of the disorder. Thus, the DSM-III-R has only slightly changed from the criteria for schizophrenia put forth in the DSM-III and these changes were only made to include the results of recent research (Skodol, 1989).

Since it has only recently been published, little research has been reported regarding the definition of schizophrenia in the DSM-III-R, its relation to the definition in the DSM-III, or its relation to other diagnostic symptoms. Nonetheless, some research is beginning to emerge. For example, Fenton, McGlashan, & Heinssen (1988, 1989) employed the DSM-III-R criteria for schizophrenia to rediagnose 182 patients who

were diagnosed originally as schizophrenic in accordance with the DSM-III criteria. Their results indicated that 10% of their sample (18 patients) were no longer diagnosed as schizophrenic. To determine what factors might differentiate these patients from those who remained classified as schizophrenic, Fenton et al. compared the two groups on 14 demographic, premorbid, and outcome variables (e.g., marital status, premorbid functioning). No significant differences were in evidence between the groups. According to Fenton et al., these results suggest that there was an even further narrowing of the definition of schizophrenia by the adoption of the DSM-III-R criteria. It should be noted, however, that their was no attempt to reclassify those patients who were diagnosed non-schizophrenic according to the DSM-III. Thus, their conclusions might be viewed as somewhat premature.

The DSM-III-R diagnosis of schizophrenia has also been the target of criticism. As with the DSM-III, there are concerns that the rapid changes in criteria may reflect the results of unreplicated research (Cooper & Michels, 1988; Gift, 1988). Also, some theorists, such as Carson (1991), felt that the criteria are a "hodge podge" and "conceptually unintegrated". Other researchers have investigated specific criteria such as the special emphasis on "bizarre delusions" and have found them to be unreliable across raters (Flaum, Arndt, & Andreasen, 1991) and not to contribute to the diagnositic replicability of the criteria (Goldman, Hien, Haas, Sweeney, & Francis, 1992). Spitzer and his colleagues recognize the disagreement with respect to the optimal definition for schizophrenia (Spitzer & Williams, 1988). As a result, they listed it as one of the "established diagnoses" which was in need of further study in the development of the DSM-IV.

Field trials are now being undertaken for DSM-IV options for schizophrenia (Flaum & Andreasen, 1991). The importance of this set of field trials is underscored by reports that only 11 diagnoses were included for such trials by the US National Institute of Mental Health. These first sets of criteria were developed after several literature searches and new examinations of unpublished data sets (cf. Andreasen, 1991). The changes reflect the views (1) that negative symptoms (i.e., the result of a deficit in normal functioning such as flat afect) are more central to the disorder than previously had been articulated in the criteria, and (2) that "bizarre" delusions had low diagnostic clinical utility (Andreasen & Flaum, 1991; Task Force on DSM-IV, 1991).

General Conclusions. In reviewing these systems, two points stand out regarding the complexity of the systems and the narrowness of the definitions. First, in comparing the systems throughout the last 30 years, they have become more and more complex. This complexity can be seen in comparing two earlier systems (FRS and DSM-II) and two of the newer systems (RDC and DSM-III-R). A more direct comparison can be seen in the changes between the DSM-II and the DSM-III. The increasing complexity of the systems is the result of their developers becoming more aware of the intricacies of the disorder, especially what specific criteria separates schizophrenia from other disorders.

This increasing recognition of the complexity of the definition has led to a narrowing of the definition's scope. As has been noted in previous research, the majority of patients who are diagnosed as schizophrenic by the narrower systems are usually also diagnosed as such by the wider systems. In addition to these patients, the wider systems include patients that the narrower systems would diagnose as

schizo-affective, paranoid, or manic (e.g., Endicott et al., 1982). Nevertheless, the question remains: Is the narrower system preferable to a wider system?

This question relates to the older debate between the proper balance of "bandwidth" and "fidelity". On one side of this debate is bandwidth. The wider the bandwidth, the more cases either of a disorder or of a type will be assured to be in the resultant group (i.e., higher true-positive number). A price is paid, however, for this increase in the number of true cases. That price is that the wider the bandwidth, the more non-pure cases of the disorder will also be included (i.e., higher false-positive number). The percentage of non-pure cases which are included increases as the bandwidth increases (higher false-positive:true-positive ratio). Thus, the fidelity of the resultant group is sacrificed at the expense of assuring that a greater number of pure cases are included. Thus, bandwidth and fidelity work against one another, such that one increases in a system only at the expense of the other.

This balance can be seen in the diagnosis of schizophrenia. As noted earlier, the DSM-III and the DSM-III-R have narrower bandwidths than most of the other systems. Yet, they are often the most predictive of future symptomatology. Thus, the patients are not suffering from a transitory set of symptoms. Instead, they seem to be displaying symptoms which result from a common underlying disorder. This forsaking of the breadth of the bandwidth in order to achieve greater diagnostic precision was an aim of the DSM-III (Millon, 1983).

As Andreasen (1987) has argued, a narrow clinical definition of schizophrenia is well-suited for patient care. It allows for the proper

treatment to be given to those patients diagnosed as schizophrenic. For example, few manics would be denied lithium treatment because they were originally diagnosed as schizophrenic. Furthermore, research with a narrow sample allows for the patient group not to be contaminated by patients with an affective disorder.

It should be noted, however, that there are problems inherent in using a narrower definition of schizophrenia. From a treatment perspective, some patients with schizophrenia will not receive the proper medication for the control of their symptoms. Furthermore, some forms of research, such as heritability research, prefer a wider range of patients. By including those disorders related to schizophrenia (e.g., affective disorder with mood incongruent delusions, schizotypal personality disorder), more variance due to genetic factors can be identified. Nonetheless, such research recognizes that while these disorders are related to schizophrenia, they are not pure cases (Farmer, McGuffin, & Gottesman, 1987).

The Subtyping of Schizophrenia

As the definition of schizophrenia became narrower, some researchers began to be concerned that it was becoming too homogeneous and could loose the richness of the heterogeneity of the disorder (e.g., Stephens et al., 1982). Nevertheless, research using the narrower diagnostic systems has continued to report consistently broad variance in test results. Such reports continue to suggest the inherent heterogeneity of schizophrenia (Andreasen, 1987).

Since the time of Kraepelin and Bleuler, schizophrenia has been considered to be composed of various subtypes, each having its own array of symptoms. While these authors made attempts at defining the

subgroups, they recognized that their attempts were only preliminary. Bleuler (1911/1950) wrote that "at the present time, we cannot solve the problem of dissecting schizophrenia into its natural subdivisions" (p. 227). In attempts to subdivide schizophrenia more adequately, numerous methods of subtyping have developed over the years. The following section describes some of the more widely used subtyping systems, reviewing their relative strengths and weaknesses.

Classic subtypes. To a substantial degree, the classic subtypes continue to be the standard for psychiatric diagnoses (McGlashan & Fenton, 1991). The types put forth by the American Psychiatric Association (1987; DSM-III-R) closely relate to those types identified by Kraepelin and Bleuler: Catatonic, Disorganized (Hebephrenic), Paranoid, Undifferentiated, and Residual. Likewise, subtypes proposed for the World Health Organization's new ICD-10 (1988) and the upcoming DSM-IV (Task Force on DSM-IV, 1991) generally parallel those in the DSM-III-R. Simple schizophrenia was not included in the DSM-III/DSM-III-R because it was seen as difficult to separate from severe personality disorders and because of the lack of evidence, at the time, that it shared some important features with the other subtypes of schizophrenia (Spitzer et al., 1978). Several researchers have argued that it should not have been excluded (e.g., Black & Boffeli, 1989; Dworkin, 1992). Initial reports indicate that it may be in an appendix in DSM-IV to allow for its further study and that it has been included in the ICD-10.

Nonetheless, support for the continued use of these classic subtypes has been wanting in several respects. For example, the rates for the diagnosis of the various subtypes has changed considerably over

the years. The percentage of patients diagnosed as suffering from hebephrenic and catatonic schizophrenia declined from 11% of admissions to the Iowa State Psychiatric Hospital in 1920 to 1% in 1966 (Morrison, 1974). Furthermore, the categorization of patients into one of these classic subtypes often is unreliable because patients often will display symptoms from more than one subtype (Hay & Forrest, 1972). The more recent attempts at employing a classically-derived subtyping, such as the DSM-III-R, have eliminated much of this criticism through the use of narrower and more well-defined diagnostic criteria. Nonetheless, the utility of the classic subtypes remains in question due to the dissatisfaction arising from their problems (Andreasen, 1987).

Statistically-Derived Subtypes. Multivariate statistical procedures have been employed for several decades as a tool for the investigation of naturally occuring subtypes within groups of psychiatric patients. Early research focussed on factor analysis (e.g., Trouten & Maxwell, 1956), which forms a classification scheme by assuming that naturally occuring dimensions of psychopathology could be identified if the appropriate relevant variables were included in the analysis. While the majority of this research encompassed several diagnostic categories, a few centred on schizophrenia (e.g., Garfield & Sundland, 1966; Nuttall & Solomon, 1965). Such research, however, was often contradictory or inconclusive (cf. Costello, 1970). As a result of these problems, enthusiasm for factor analysis slowly faded away and the technique was rarely used for such purposes beyond 1970.

Nonetheless, some factor analysis research has continued. For example, Liddle (1987) assessed 40 chronic DSM-III-diagnosed patients with measures of a wide range of symptomatology. He performed a factor

analysis which supported three factors with each having high loadings for separate groups of symptoms. These three "syndromes" were (1) a psychomotor poverty syndrome, (2) a disorganization syndrome, and (3) a reality distortion syndrome. Liddle and Barnes (1990) have since replicated these three factors with a separate group of 57 chronic DSM-III schizophrenics. This research did replicate these patterns among symptoms. It failed to support, however, the subsequent subgroupings of these patients by the three "syndromes".

The attention of many researchers has shifted to the use of cluster analysis. This technique has been employed periodically during the last 20 years in an attempt to discover empirically-based subtypes within schizophrenia (e.g., Carpenter, Bartko, Carpenter, & Struass, 1976; Farmer, McGuffin, & Spitznagel, 1983). Cluster analysis is a multivariate statistical method that can be used to identify consistent profiles of symptoms from a large number of subjects (i.e., those sharing similar profiles are "clustered" together; cf. Corning & Steffy, 1979). Early research employed samples of psychiatric patients with widely divergent diagnoses in attempts to determine the natural typology of classification. For example, Everitt, Gourlay, and Kendell (1971) employed 70 "mental state" and "history" items derived from a sample of 236 American and 244 British psychiatric patients. Their sample included over a dozen separate diagnostic groups (e.g., residual schizophrenia, manic-depressive illness, drug dependence) and the aim was to validate these categories. Everitt et al. reported on 11 clusters and four of these clusters might be interpretted as possible subtypes of schizophenia: "General factor of (paranoid) schizophrenia", "Depressive delusions", "Schizoid personality / schizophrenic defect

state", and "Auditory hallucinations". While the authors believed these subtypes were meaningful and useful, no research has since been attempted to investigate or replicate them.

Such research has also been employed solely on groups of patients diagnosed as schizophrenic in attempts to determine subtypes. For example, Carpenter, Bartko, Carpenter, and Strauss (1976) used 27 symptoms from the Present State Examination to divide 600 schizophrenics into four distinguishable and clinically meaningful subtypes. In another example, Farmer et al. (1983) employed two different cluster analysis algorithms on a list of 42 variables derived from 76 schizophrenics. Both of these programs resulted in the definition of two reasonably distinct subtypes. Patients in the first subtype were characterized by delusions, better premorbid adjustment, and a later age of onset. The patients in the second cluster were characterized by a family history of schizophrenia, incoherent speech, blunted affect, and auditory hallucinations. Just as in Everitt et al. (1971), however, there has been no further research on these subtyping schemes.

In reviewing the research employing cluster analysis in psychiatric classification, Skinner and Blashfield (1982) concluded that "to date, cluster-analytic techniques have had limited impact on psychiatric nomenclature, and clinicians have not found the cluster-derived syndromes to be relevant to everyday practice" (p. 727). Meehl (1979) took an even more extreme position, stating that the cluster algorithms have not been responsible for the discovery of any single taxon in psychopathology, or even in organic medicine. In their review of this topic, Spitzer and Williams (1985) were able to identify only one

addition to one classification schema which resulted from such analyses (DSM-II's "Runaway reaction of childhood"). They concluded that apart from this addition, "no category has been added to a classification of mental disorders for clinical use that was first identified by a mathematical procedure designed to generate diagnostic categories" (p. 601). The factors underlying this minimal impact were outlined by Skinner and Blashfield as (1) a marked contrast between clustering research and the traditional approach for identifying syndromes in clinical medicine, (2) the increase in the complexity of the empirically-derived subtypes makes them of little value in communicating information among medical professionals, (3) little extended research with empirical subtypes, and (4) difficulties in the different methodologies of clustering employed by different researchers. The authors concluded, as have other theorists (e.g., Millon, 1991), that while cluster analysis may be useful empirically in the future, it will make little lasting impact until the need for clinical acceptance of the resulting subtypes is addressed.

Biological Subtypes. Many researchers propose that the reason that the results of biological research into schizophrenia are inconclusive is that it is a biologically heterogeneous disorder (Meltzer, 1979). This heterogeneity then results in considerable individual differences among schizophrenics on these measures (Wolkowitz, Bartko, & Pickar, 1990). By investigating the symptoms presented by schizophrenics with and without certain proposed biological abnormalities (e.g., enlarged ventricles, DA abnormalities), it has been hoped that new specific subgroups can be outlined (see below).

Yet, just as there is difficulty in the definition of schizophrenia from a biological basis (cf., Karson et al., 1986), biological subtyping has been unsuccessful. One very good example of this approach is illustrated in a review by Wyatt, Potkin, Kleinman, Weinberger, Luchins, and Jeste (1981) of attempts to discriminate possible biological subtypes of schizophrenic patients. The authors reviewed six measures (platelet MAO activity, phenylethylamine concentration, brain NE concentration, abnormalities on CT, lateralization asymmetries, and the presence or absence of tardive dyskinesia) and concluded that all methods were able to successfully subtype patients. Wyatt et al. also concluded, however, that it was difficult to determine what was the cause of the differences: the disorder itself or a by-product of the disorder. Furthermore, they concluded that the subclassification schemes from biological measures were rarely confirmed by any further research. While research continues in biological subtyping (e.g., Davila et al., 1989), there have been few solid advances.

Positive and Negative Subtypes. The most well known of these proposed divisions is Crow's (1980a, 1980b) Type I/Type II hypothesis. Crow put forth the argument that there are two distinguishable forms of schizophrenia: Type I and Type II. The basis for the classification of these two subtypes was the view that there were two separate clusters of symptoms which had differential reactions to treatment and different courses (see Table 2). One group of symptoms, defining Type I, were positive: that is, these symptoms were additional to an individual's normal functioning (e.g., hallucinations). The second group of Type II symptoms were classified as negative because they were the result of deficits in normal functioning (e.g., flat affect). These two subtypes

Table 2

Type I and Type II Syndromes of Schizophrenia

	Type I	Type II
Symptoms	Positive symptoms - delusions - hallucinations - thought disorder	Negative symptoms - flat affective - poverty of speech - loss of volition
Type of illness in which most commonly seen	Acute schizoprhenia	Chronic schizophrenia
Potential for response to neuroleptics	Good	Poor
Presence of intellectual impairment	Absent	Sometimes present
Outcome	Reversible	? Irreversible
Pathological process	Increased dopamine receptors	Cell loss and structural changes in the brain

Based on Crow (1980a).

were then employed to interpret previous research results, such as differential response to neuroleptics. While the view that schizophrenic symptoms can be divided into positive and negative symptoms is not new (cf., Berrios, 1985), the view that they were actually the result of two different forms of schizophrenia was novel and provoked considerable discussion and research.

The research which resulted, however, has not been supportive of Crow's Type I-Type II dichotomy. First, research has not been able to separate patients into the two subtypes as a result of the two types of symptoms (Rosen et al., 1984). A second point of difficulty was that often what one researcher thought of as a negative symptom was defined as a positive symptom in another study. For example, this lack of strong definitions as to what is negative and what is positive is evident in research on thought disorder. Crow (1980a) originally viewed thought disorder as a positive symptom but subsequent research defines many aspects of thought disorder (e.g., difficulty in abstract thinking, stereotyped thinking) as negative symptoms (Lindenmayer, Kay, & Opler, 1984). Third, the utility of the dichotomy of positive and negative symptoms of schizophrenia has been questioned. For example, Lindenmayer, Kay, and Friedman (1986) found that the negative symptoms of schizophrenia were difficult to separate from the symptoms of depression. For these reasons, among others, the sharp distinction of the two subtypes has been criticized (cf., Andreasen, 1985; Sommers, 1985).

Even critics of Crow's theory, however, grant it some merit. This theory renewed interest in the concepts of positive and negative symptomatology (Andreasen, 1987). This interest has led to the

development of scales specifically to measure positive and negative symptoms (cf. Fenton & McGlashan, 1992) along with theories aimed at elucidating the division (cf., Walker, 1987). Also, there has been a significant increase in the recognition of the importance of negative symptoms in schizophrenia (cf., Barnes, 1989). Nevertheless, the lack of consistent research support has challenged the claimed effectiveness of the Type I/Type II syndromes as descriptors of schizophrenia. As a National Institute of Mental Health (NIMH) panel concluded, after reviewing the present state of research and theory of the clinical phenomenology of schizophrenia, "while heuristic and hypothesis-generating, (the Type I/Type II dichotomy) was an oversimplification" (p. 351; Andreasen, Shore et al., 1988).

Paranoid and nonparanoid subtypes. While the utility of employing the four classic subtypes has been questioned (Andreasen, 1987), one of those subtypes has remained viable: paranoid schizophrenia. Paranoid schizophrenia differs in many respects from the other forms of schizophrenia (McGlashan & Fenton, 1991; Skodol, 1989). For example, Morrison (1974) reviewed the records of admissions to a state psychiatric hospital over a period of 46 years. He discovered that although the frequency of schizophrenia remained constant, there was considerable variability from year to year in the frequency of the diagnosis for most subtypes. The exception was that the percentage of diagnoses of paranoid schizophrenia remained relatively constant. Morrison viewed such stability in diagnostic incidence as support for the division of schizophrenia into broad paranoid and nonparanoid subtypes. He believed that any further subdivision of nonparanoid

schizophrenia was susceptible to either environmental influences or diagnostic fads.

Further support for this subdivision can be found in the WHO's (1973) International Pilot Study of Schizophrenia. In this study, the rates of the diagnoses of the subtypes of schizophrenia in nine different countries were compared. The only subtype that was measured reliably across different centres was paranoid schizophrenia. The symptom profiles in eight centres showed a significant degree of concordance with one another. The other centre, in the United States, had a profile that was significantly in concordance with four of the other eight centres. Other subtypes were either not diagnosed with sufficient frequency or the profiles were not as frequently in concordance. Research continues to indicate that the patients from these two broad subtypes differ in many respects (cf. McGlashan & Fenton, 1991; Nicholson & Neufeld, in press).

To take one example, the courses of the two subtypes differ considerably. Paranoid schizophrenia on the whole may be less disruptive than nonparanoid schizophrenia. Evidence suggests that individuals with paranoid schizophrenia tend to develop their disorder at a later age and tend toward better premorbid adjustment (e.g., a higher incidence of marriage, higher social competency) than those individuals with nonparanoid schizophrenia (Burack & Zigler, 1989). Likewise, while inpatients they appear to be more socially competent when compared to inpatients with nonparanoid schizophrenia (Dobson & Neufeld, 1987). Following diagnosis, paranoid symptoms are reduced faster and to a greater degree than nonparanoid symptoms (Goldberg, Schooler, & Mattsson, 1967). Thus, paranoid schizophrenics have briefer

hospitalizations and experience fewer rehospitalizations (Strauss, Sirotkin, & Griswell, 1974). Finally, numerous studies have indicated that the outcome for individuals diagnosed with paranoid schizophrenia is generally better than the outcome for nonparanoids (e.g., Kendler, Gruenberg, & Tsuang, 1984). Taken together, this body of research indicates that considerable clinically-relevant differences exist between the two subtypes of patients and the course of the disorder.

Support for this classification can be found in a variety of studies. The previously discussed cluster analysis research is one instance. For example, the two clusters resulting from Farmer, McGuffin, and Spitznagel's (1983) analysis partially parallel the paranoid/nonparanoid division. Patients in the first cluster were characterized by delusions, better premorbid adjustment, and a later age of onset. Those patients in the second cluster were characterized by a family history of schizophrenia, incoherent speech, blunted affect, and auditory hallucinations. Because of these parallels, Farmer et al. argued that two clusters reasonably may be considered paranoid and hebephrenic/nonparanoid subtypes.

In addition, a small body of biochemical research tentatively supports the paranoid/nonparanoid dichotomy (e.g., platelet serotonin levels; Muck-Seler, Jakovljevic, & Dennovic, 1991). Following a meta-analysis of studies of platelet monoamine oxidase (MAO) activity among paranoid and nonparanoid schizophrenics, Zureick and Meltzer (1988) concluded that the typical paranoid schizophrenic did have lower activity than 61% of nonparanoid schizophrenics and 71% of normal controls. They concluded also that these differences were not the result of medication, demographic, diagnostic, or methodological

factors. Thus, there is some biological evidence for the division of schizophrenic patients into these two subtypes.

Other fields of experimental research have reported consistent paranoid/nonparanoid schizophrenic differentiae. Laboratory studies of cognition compose one such field. Encouraged by Chapman and Chapman's (1973) review of differences between paranoid and nonparanoid schizophrenics on a large number of laboratory tasks, researchers have continued to document performance distinctiveness, and have theorized about their sources (e.g., Broga & Neufeld, 1981; George & Neufeld, 1987; Magaro, 1981). It has been suggested that paranoid schizophrenics have particular difficulty in extracting from their environment the stimulus properties necessary for "informed responses". Specifically, samples of these patients appear quite consistently to be slower in cognitively translating ("encoding") relevant stimuli into a format that facilitates subsequent operations taking place in "working memory". It is in these operations that the appropriate response is identified vis a vis existing task requirements. Nevertheless, while "encoding" is slowed, the subsequent operations in "working-memory" remain intact (Neufeld, 1991; Neufeld, Vollick, & Highgate-Maynard, in press).

An example of experimental cognitive research is Broga and Neufeld's (1981) study of cognitive performance and response styles in schizophrenics. Forty nonpatients, 20 paranoid schizophrenics, and 20 nonparanoid schizophrenics had a battery of seven standard experimental psychology tasks (e.g., Sternberg Choice Reaction Time Task, digit span) administered to them. From these seven tasks, seventeen constituent measures were available. A multivariate analysis of these measures found two significant discriminant functions for the

separation of the three groups. The first function, which accounted for 63% of the between group discrimination, reflected processing efficiency. It maximally separated paranoid schizophrenics and nonpatients with the nonparanoid schizophrenics falling between these two groups.

The second function, which accounted for the remaining between group discrimination, suggested differential response style. That is, on one end of the function there was a tendency to protect against false negatives by the patient emitting a response lest he or she miss a correct answer. On the other end of this second function, there was a tendency for protection against false positives. Thus, patients on this end of the function would tend to withhold a response to a task rather than take the chance of making an incorrect response. This second function maximally separated paranoid and nonparanoid schizophrenics with nonpatients falling in between these two subtypes.

In summarizing these discriminant functions, Broga and Neufeld characterized paranoids as drawing "certain inferences more liberally from presenting stimulation despite processing it less effectively" (p. 506). Nonparanoids, on the other hand, "tended to process more effectively than paranoids but were more conservative in drawing certain inferences" (p. 506). While cautioning against overinterpreting their results, the bidimensional characterizations of the schizophrenic samples are in keeping with the clinical symptomatology of the patient subtypes.

Another example of recent research which highlights differences between paranoid and nonparanoid schizophrenics was conducted by George and Neufeld (1987). In reviewing the previous research on differences

in hemispheric deficits between the paranoid and nonparanoid subtypes, George and Neufeld found an undesirable level of inconsistency. They proposed that the differences could have been due to differing amounts of "attentional resources" necessary in the tasks in the different studies. In their research, five subject groups (paranoid schizophrenics, nonparanoid schizophrenics, nonschizophrenic psychiatric in-patients, normal controls recruited from the city at large, and university students) took part in a battery of eight tachistoscope tasks. Different amounts of processing were required for the eight tasks. The results indicated that the psychiatric groups were generally slower than the control groups in their responses, reflecting a generalized decrease in ability to utilize "attentional resources". There was also evidence that the nonparanoids displayed the greatest evidence for a generalized deficit in available resources from attentional pools. Their results also indicated that the inconsistency of previous research into paranoid-nonparanoid differences in hemispheric deficits could have been the result of differing amounts of processing load employed by the tasks in these different studies. Both this study and Broga and Neufeld (1981) indicate how the distinction between the paranoid and the nonparanoid subtypes have been paralleled by distinct performance patterns on laboratory tasks.

Even though patients designated as paranoid versus nonparanoid schizophrenic apparently are separable on measures such as those described above, concerns have been raised about the "arbitrariness of the designation". Definitional problems always have been present in the diagnosis of schizophrenia and other forms of psychopathology (cf., Klerman, 1986). Problems in the assignment of paranoid and

nonparanoid status have been noted for a number of years (e.g., Berkowitz, 1981; Katz, Cole, & Lowery, 1964). As a result, simple statements about the exact nature of these two subtypes of schizophrenia are elusive.

Predominant symptoms of alternate subtypes, of course, may not be mutually exclusive. In their investigation of the Maine Paranoid Scale (a measure designed specifically to separate schizophrenics into paranoid and nonparanoid categories), Magaro, Abrams, and Cantrell (1981) noted the difficulty of symptom overlap inherent in such categorization. To overcome this problem, Magaro et al. suggested that patients should be recorded as "unclassifiable" when they do not clearly display one set of symptoms over the other. In the DSM-III-R, a patient with both types of symptoms would be classified as "Undifferentiated Type". This difficulty in the lack of a firm border between the two categories is not limited to paranoid/nonparanoid subtyping (cf. Crow's Type I/Type II hypothesis). Nonetheless, it is a significant problem which persists within approaches to the paranoid/nonparanoid distinction.

The plot thickens further when change in symptomatology over time is brought into play. Generally, paranoid symptoms have been shown to decline with the passage of time, and often disappear (Bridge, Cannon, & Wyatt, 1978; Depue & Woodburn, 1975). Furthermore, there is strong evidence that with chronicity, paranoid schizophrenics slowly lose their paranoid symptoms and develop into "nonparanoid schizophrenics" (Pfohl & Winokur, 1983; Ritzler, 1981). While there are cases of changes in the opposite direction, they are considerably less common (Tsuang, Woolson,

Winokur, & Crowe, 1981). Thus, imputing subtype membership is qualified additionally by the risk of symptom instability.

Focusing on points of divergence rather then overlap, some theorists have hypothesized that two independent disorders or processes give rise to the symptom patterns. Meissner (1981) and Magaro (1981) both hypothesized a schizophrenia separate from a paranoid process. Magaro's (1981) categories, however, differ from those processes put forth by Meissner in the definition of paranoid schizophrenia. Meissner hypothesized that it is when the two processes are combined that there is evidence for the existence of paranoid schizophrenia. On the other hand, Magaro viewed "paranoid schizophrenia" as an extreme form of the "paranoid process". It was, according to Magaro, independent of the schizophrenic process. Similar to this view is the proposal by Zigler and Glick (1984, 1988) that paranoia is a mechanism for coping with depression. As a result, some varieties of paranoid schizophrenia might be viewed as extreme examples of "camouflaged depression". By providing their own theories, these various authors attempted to overcome some of the difficulties inherent in the standard division of schizophrenia into paranoid and nonparanoid subtypes.

The present formulation addresses continuing concerns regarding the natural course of paranoid and nonparanoid forms of schizophrenia. For example, if the processes are independent, why should chronic paranoid schizophrenics often develop nonparanoid symptomatology and have their paranoid symptomatology diminish? Further, formulating two theoretically independent processes may downplay unnecessarily bona fide points of linkage that brought the two symptomatologies together in the clinical literature (Clementz & Sweeney, 1989).

Proposed Reconceptualization of the Paranoid - Nonparanoid Distinction

An Overview. In an effort to overcome some of the difficulties concerning the division of schizophrenia into paranoid and nonparanoid subtypes, a reconceptualization of the relation between the observable symptoms to the "latent disorder" is proposed. Originally outlined by Nicholson and Neufeld (in press), this proposal includes two independent continuous factors: (1) severity of disorder and (2) severity of symptoms. The first factor, severity of disorder, posits paranoid schizophrenia to be a milder form of the disorder and nonparanoid schizophrenia to be a more severe form. Thus, the type of symptoms displayed are indicative of the severity of disorder from which they emanate.

The second factor, severity of displayed symptoms, is independent of the type of displayed symptoms. Severity of symptoms concerns their relative frequency and prominence in one's behavioral repertoire. Symptoms appearing more or less often during a given epoch may follow a course of shorter or longer persistence over an individual's life; may be combined with better or worse premorbid social competence; and may accompany varying pervasiveness of problems in cognitive functioning. The same can be said regarding symptom salience. Severity of disruption of "normal development and routine functioning" increases with both severity of disorder and severity of symptoms.

The proposed formulation will be elaborated upon, with reference to Figure 1. In the figure, the factors create a two-dimensional plane of disorder. The first factor, severity of disorder, ranges from "Paranoid' to "Nonparanoid". The second factor ranges from a level of "Absence" of symptoms to the "Most severe" of displayed symptoms.

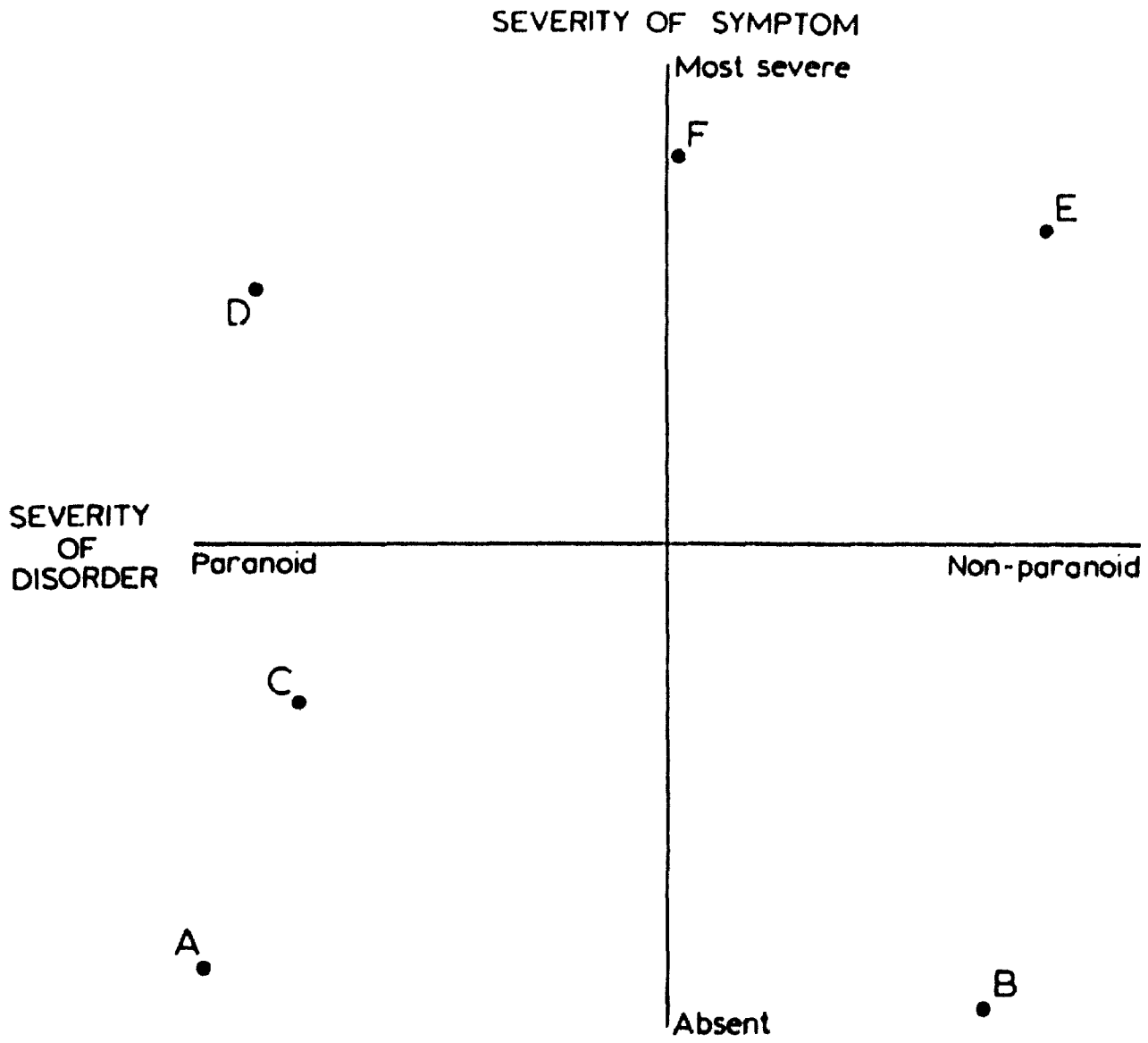
FIGURE 1

A two-factor model of schizophrenia:

Severity of symptom and severity of disorder

(Hypothetical patients location in the

two-dimensional plane as points A Through F)


SEVERITY OF SYMPTOM
Most severe
F
E
D
SEVERITY
OF
DISORDER
Paranoid
Non-paranoid
C
A
B
Absent

Patients placed at points A and B in the field would be relatively symptom-free. If Patient A were ever to develop symptoms, however, he or she would develop paranoid symptoms (e.g., bizarre delusions; cf. Meehl, 1962, 1989, 1990; Nicholson & Neufeld, 1992). Patient B, on the other hand, would be more prone to develop non-paranoid symptoms (e.g., prominent hallucinations, flat or inappropriate affect). Furthermore, these types of symptoms might already be present in Patients A and B but at benign levels. Patient C would display some mild paranoid symptoms. These symptoms could be at the level of a mildly paranoic personality or, perhaps, a paranoid personality disorder. The patient at point D in the field would be classified as a paranoid schizophrenic patient, displaying severe symptoms of the less severe form of the disorder. Any patient at point E would display a severe level of symptoms of the severe form of the disorder. Such a patient would then be classified as a nonparanoid schizophrenic patient. Patient F displays high levels of a mixture of paranoid and nonparanoid symptoms. This "mixed" case is difficult to classify under the more standard systems. A classification of Patient F as "paranoid schizophrenic" would bypass much of his or her manifest nonparanoid symptomatology and result in an overattribution to Patient F of paranoid symptomatology.

Severity of Disorder. As noted above, this continuum of disorder locates paranoid and nonparanoid schizophrenia toward the milder and more severe poles, respectively. The type of symptoms that an individual displays would reflect the placement he or she will have with respect to these positions (Figure 1 - D and E). Thus, defining the two subtypes as the extremes of a single continuum accomodates differing

symptoms linked to the same disorder, then, according to the disorder's severity.

The validity of this stance is considered from several perspectives. First is its compatability with empirical distributions among patients of paranoid and nonparanoid symptomatology, including distributions obtained from cluster analysis and multidimensional scaling research. Second, genetic research on the subtypes of schizophrenia will be reviewed to show their consistency with a continuum of severity model. Third, the relative merits of continuum approaches in related areas of psychopathology are considered. Finally, the strengths and weaknesses of continuum approaches are reviewed.

The proposed linkage of the two subtypes of schizophrenia by a continuum of disorder severity broaches certain enigmas in the topography of symptom occurence and co-occurence, as follows. First and foremost, it allows for an individual to display both paranoid and nonparanoid symptoms (i.e., the symptoms from both ends of the continuum; Figure 1 - F). Such individuals otherwise would be unclassifiable or viewed as "mixed" (e.g., "Undifferentiated" in DSM-III-R). They now could be seen as located toward the centre of the continuum.

Second, as the disorder becomes more chronic and the patient deteriorates, the paranoid schizophrenic patient could display the symptoms representative of a growing severity of his or her disorder. In other words, his or her paranoid symptoms could gradually lessen in severity and he or she could develop the symptoms indicative of the more severe (i.e., nonparanoid) form of schizophrenia. Since it would be rare that a chronic patient would develop a less severe form of the

disorder, it is also rare that the symptoms displayed by a schizophrenic patient would change from nonparanoid to paranoid.

Third, this continuum approach "allows" some individuals initially to display one extreme form of schizophrenia and others to display the other form. The approach complies with a view put forth by Cromwell (1975) and by Zigler, Levine, and Zigler (1976) that the putative milder form, paranoid schizophrenia, is developmentally associated with a more advanced level of maturity. It would be expected, then, that paranoid symptomatology might have a later age of onset, preceded by better premorbid status. Finally, a better prognosis should follow from a milder form of disorder. The disorder-severity continuum, then, appears consistent with empirical observations on symptom mixture at a given time, symptom transition over time, relations of symptoms to social competence and age of onset, as well as differential prognosis.

Fourth, this continuum is also consistent with research that paranoid schizophrenics are less impaired than nonparanoid schizophrenics on a variety of measures. For example, clinical studies have indicated that nonparanoid schizophrenics may perform more poorly then paranoid schizophrenics on some neuropsychological measures (Bornstein et al., 1990; Levin, Yurgelun-Todd, & Craft, 1989). Evidence supporting such differences have been described on a number of measures with the greatest amount of recent interest in perseveration errors on the Wisconsin Card Sorting Test (e.g., Rosse, Schwartz, Mastropaolo, Goldberg, & Deutsch, 1991).

Evidence for a continuum model can also be seen in research on the genetics of schizophrenia subtyping. Studies generally have indicated that the relatives of paranoid schizophrenics have a lower rate of

schizophrenia than do the relatives of nonparanoid schizophrenics (e.g., Kallman, 1938; Onstad, Skre, Torgerson, & Kringlen, 1991; although this finding is not always replicated e.g., Kendler, Gruenberg, & Tsuang, 1988). As a result, theorists such as Rosenthal (1970) have suggested that the paranoid subtype will be present when there is a fewer number of "pathological genes". Hebephrenic or catatonic subtypes were to be present in those individuals who have the most of these genes.

The present model is consistent with this approach in that more genetic influence would be evident in the more severe (i.e., more nonparanoid) schizophrenia (e.g., Pogue-Geile & Harrow, 1985). Such a continuum model is consistent with multifactorial polygenetic models (Farmer, McGuffin, & Gottesman, 1990; Fowles, 1992; McGuffin, Farmer, & Gottesman, 198?; Tsuang, Lyons, & Faraone, 1990). It could also be viewed as consistent with a single gene model that included several environmental factors and polygenic potentiating factors (cf. Meehl, 1990). It is interesting to note that research has indicated also that non-schizophrenic patients who have been diagnosed as having a disorder with "schizophrenic" symptoms (e.g., atypical psychoses, affective disorder with mood-incongruent delusions) have demonstrated a genetic relatedness to schizophrenia (Baron & Gruen, 1991; McGuffin et al., 1987). According to the present two-factor model, it is reasonable that such individuals would display symptoms at a level lower than that necessary for a diagnosis of schizophrenia and/or have a co-morbid disorder that disallows a diagnosis of schizophrenia (according to current diagnostic categories).

Several models have been developed to combine such genetic continuum models with symptomatology (e.g., Gottesman, 1991; Nuechterlein, 1987). Such models are necessary to explain how differences in symptomatology and course of disorder can exist in genetically identical individuals (cf. the Genain Quadruplets; DeLisi et al., 1984). For example, Nicholson and Neufeld's (1992; Neufeld and Nicholson, 1991) Dynamic Vulnerability Formulation on stress and schizophrenia was proposed as an extension of previous genetic vulnerability-environmental stress models. This model suggests that genetic endowment affects an individual's vulnerability to the disorder as well as his or her stress-appraisal and coping mechanisms. The displayed symptomatolgy both is affected by genetic endowment and coping mechanisms and affects stressors, appraisal mechanisms, and coping mechanisms. The dynamic interplay of these factors over time results in changes both in symptom form and in the severity of those symptoms. Models such as the Dynamic Vulnerability Formulation are consistent both with the continuum models of genetic influence on schizoprhenia and with the proposed two-factor model.

How might paranoid and nonparanoid symptomatology be distributed according to cluster analysis results? The possibility of continuity was suggested by Farmer et al. (1983) in their study of 42 signs and symptoms that resulted in a two-cluster solution. The first cluster was characterized by delusions, better premorbid adjustment, and a later age of onset. The second cluster tended to have a family history of schizophrenia, incoherent speech, blunted affect, and auditory hallucinations. Thus, as stated earlier, Farmer et al.'s solution closely paralleled the paranoid/nonparanoid distinction.

Farmer et al. observed that the signs and symptoms discriminating the two clusters tended to be present among the second cluster of patients and absent from the first. The exceptions were the paranoid symptoms such as "well-organized delusional system". They stated, therefore, that the results "...indicate that we have not shown separate entities but our cases fall on a mild-severe continuum, with Cluster 2 (the nonparanoid cluster) representing the more severe form of the illness" (p. 6). The authors noted that cluster analysis methods do not differentiate between continuous and discontinuous distributions in fulfilling the group formation criteria embodied in clustering algorithms. They argued, therefore, that their analysis might have divided a symptomatologically continuous patient sample into distinct groupings artifactually.

This possibility was examined by Farmer et al. and rejected as follows. They submitted bona fide patient profiles, and randomly generated profiles of signs and symptoms, to each of two cluster algorithmic procedures. The co-assignment of patients to the same group by the procedures was greater when real patient data was used than when random data was used. This finding, they argued, indicated that the categorical groupings were not "arbitrary or artifactual" and, therefore, the schizophrenic patient group was not a single homogeneous cluster.

Note, however, that the above results do not contraindicate a distribution of symptomatology reflecting a mild-severe continuum of disorder. First, it can be shown that, other things being equal, the separation of clusters constructed from essentially a continuous sample will be enhanced with the use of bona fide patient data compared to

randomly generated profiles. As Meehl (1990) pointed out: "If a collection of quantitative variables is correlated, for whatever reason, dichotomizing on any of them will, given adequate statistical power, result in significant differences on those variables that are treated as dependent" (p. 78; italics in original). Profiles reflecting reduced disorder severity should tend to correlate, across measures, positively with one another, and negatively with profiles reflecting greater disorder severity. Within levels of relative severity, profiles should tend to correlate positively with one another (witness the distributions reported by Farmer et al., above). Therefore, based on the mechanics of a cluster analysis of "Euclidean distance among subjects" (the procedure used by Farmer et al.), such patterns of data will facilitate both the reduced separation among subjects within groups and the increased separation among subjects between groups (see, e.g., Neufeld, 1977).

In turn, an increase in group separation for each of two clustering solutions can be shown to facilitate agreement between the solutions with respect to subject composition of the two groups. This tendency toward increased agreement is analagous to the following psychometric observation: the correlation between two measures is increased as their correlations with a given source of variation ("common factor" or "latent variable") increase. In the present instance, the "source of variation" is the mild-severe disorder continuum; the enhanced group separation straightway represents the increased correlation of the "clustering-algorithm measures" with this source of variation; and the increased correlation between the two measures is seen in the heightened correspondence between the two algorithms in terms of the subjects who are placed together, and separated from others. All in all, if there

were a continuum and not two homogenous clusters, the clustering algorithms should still agree to the extent that Farmer et al. reported.

Research employing multidimensional scaling can also be interpreted as supporting a paranoid-nonparanoid continuum. Mirowsky and Ross (1988) investigated their theory that symptoms of any disorder would tend to cluster together but that these clusters were not clearly separated from one another. Mirowsky and Ross conducted a community survey of 463 people in two separate cities (1 in Mexico and 1 in Texas). They developed a survey for this study that measured 91 symptoms. The authors made the strong point of stating that these were "the symptoms on which psychiatric diagnosis is based" (p. 41). The items were taken from such tests as the Centre for Epidemiologic Research - Depression Scale (CES-D), the Schedules for Affective Disorders and Schizophrenia (SADS), and the Diagnostic Interview Schedule (DIS). For each symptom, the people were rated on a five-point scale as to how often it was experienced in the last 12 months. Mirowsky and Ross correlated each pair of these 91 symptoms and then employed multidimensional scaling to map the symptoms according to the pattern resulting from these 4095 correlations.

Their results, illustrated in Figure 2, indicated a pattern which they described as "like a spectrum with the ends connected" (p. 44). Mirowsky and Ross viewed it as similar to a colour wheel. When the paranoid and nonparanoid symptoms are reviewed, the pattern they illustrate is similar to that proposed by the theory. At one end of this part of the Mirowsky and Ross continuum, paranoid symptoms predominate. At the other end, nonparanoid symptoms are present. Between the two extremes, there is a mixed (i.e., DSM-III-R

FIGURE 2

Multidimensional scaling of 91 psychiatric symptoms

(p. 45; Mirowsky & Ross, 1989)

Social

Emotions

Physical

Anxiety/Mood Depression/Mania

Psychosis

"Undifferentiated") area where both paranoid and nonparanoid forms of symptomatology are present.

Continuum approaches have been proposed for several years in other areas of symptomatology (e.g., Hempel, 1961). While traditional categorization continues to be endorsed by many theorists (e.g., Klerman, 1989), others suggest that there is little evidence to support it as the most accurate representation of psychopathology (e.g., Carson, 1991; Widiger & Trull, 1991). A recent special issue of the Journal of Abnormal Psychology entitled "Diagnoses, dimensions, and DSM-IV: The science of classification" (Barlow, 1991) underscores the increasing emphasis on dimensional approaches to psychopathology. Even the DSM-III-R includes dimensional approaches: Axis IV ("Severity of Psychosocial Stressors") and Axis V ("Global Assessment of Functioning"). Nonetheless, Axis I ("Clinical Syndromes") and Axis II ("Developmental Disorders/Personality Disorders") continue to reflect a categorical approach. In its introduction, however, the DSM-III-R stated that there "...is no assumption that each mental disorder is a discrete entity with sharp boundaries (discontinuity) between it and other mental disorders, or between it and no mental disorder" (p. xxii). In some instances, the inclusion of discontinuous categories is justified primarily "...by the clinical usefulness of the distinction" (p. xxii). Such dimensional approaches have been employed by researchers to investigate disorders as varied as dyslexia (Shaywitz, Ecobar, Shaywitz, Fletcher, & Makuch, 1992), attention-deficit hyperactive disorder (ADHD; August & Garfinkel, 1989), depression (Kendell, 1969), schizoaffective disorder (Yasamy, 1987), obsessive-compulsive disorder (Salzman & Thaler, 1981), panic attacks

(Norton, Cox, & Malan, 1992), bipolar disorder (Blacker & Tsuang, 1992), and borderline personality disorder (Trull, Widiger, & Guthrie, 1990). Similar models have also been described for physical disorders such as headaches (Sheftell, 1992), Type II diabetes (Yudkin, Alberti, McClarty, & Swai, 1990), coronary artery disease (Miller, Turner, Tindale, & Posavac, 1988), and respiratory failure (Kraus, Wagner, & Draper, 1984). Some theorists have suggested that such models might be appropriate for schizophrenia but have not developed systematic models (Hafner, 1987; Weiss, 1989). In other words, there is an increasing tendency to accept a dimensional approach for some forms of psychopathology, especially with reference to personality disorders (Huemann & Morey, 1990, Livesley & Jackson, 1992; Schwartz, 1991).

The comparitive advantages of categorical and dimensional models of psychopathology have been reviewed increasingly in recent years (Feighner & Herbstein, 1987; Millon, 1991; Mirowsky & Ross, 1989; Rutter & Tuma, 1 .., Widiger & Trull, 1991). Several unique advantages have been discussed for dimensional diagnostic approaches when compared to the traditional categorical approach. Lorr (1986) listed four such advantages to a dimensional approach. First, there are no mixed, atypical, or undiagnosed cases since such cases are located in a p-dimensional attribute space. Second, cut-off scores for nondisordered/disordered decisions could be altered according to practical constraints and not based upon inflexible category boundaries. Third, a dimensional approach is superior for evaluating change since it measures degrees of change and not the simple crossing of category boundaries. Fourth, dimensional approaches repeatedly have been demonstrated to be more efficient in the prediction of patient outcome.

Likewise, in a comparison of these two approaches for the diagnosis of personality disorders, Frances (1982) discussed three advantages to a dimensional system. First, a dimensional system is superior in its precision and reliability in describing most symptoms of psychopathology. Second, such a system reduces the "halo effect" (i.e., the tendency to see behaviors in a stereotypical manner is lessened). Third, dimensional systems allow for easier manipulation of large data sets with numerous variables.

Frances also listed three disadvantages to a dimensional approach when compared to a categorical approach. First, a dimensional system tends to be weak in its attempt quickly to describe a set of symptoms. A second related problem is that the information available from a dimensional system often is complex and unwieldy. Third, there is a chance that the continuum from a dimensional approach may mask an actual underlying discontinuity.

Critics of the dimensional approach have also raised other difficulties with the approach. For example, the traditional approaches are seen as familiar to clinicians and consistent with their "disease-model" practice and their training in psychopathology (Klerman, 1989). These traditional approaches are viewed similarly by some clinical researchers who see a strong need for increased "pragmatics" and decreased "unnecessary complexity" (Feighner & Herbstein, 1987). In reviewing these arguments, Blashfield and Livesley (1991) have suggested that the dimensional/categorical argument may be becoming more a political issue with the psychiatrists on the biological categorical side and the psychologists on the continuum dimensional side of the debate. Thus, while a dimensional approach is not to be included for

Axis I and Axis II disorders in the DSM-IV, because it is believed that there is not sufficient consensus on the approach, it has now been raised to the level of a "conundrum" for the DSM-IV developers which had to be discussed seriously (Frances, First et al., 1991).

It should be stressed that employing continua of psychopathology would not pre-empt the classification of patients into categories (Achenbach, 1988; Millon, 1991). Several advantages to such a "further subdivision" have been outlined by Meehl and Golden (1982). For example, such classification makes for convenience in communication among professionals by allowing presentation of considerable patient information in summary form. Also, Meehl and Golden describe the case where such a subdivision allows for a better theoretical understanding of patient groups. By homogenizing subtypes of schizophrenic patients, a greater understanding is available of what is uniquely happening with the one group compared to another. The aforementioned research on "paranoid/nonparanoid schizophrenic subgroups" exemplifies this advantage. Such advantages tend to be forfeited when a continuum framework is placed on schizophrenic symptomatology. Nevertheless, if opting to form homogeneous subtypes, the original underlying continuum should not forgotten.

As dimensional approaches become more accepted, there is increasing debate about their merits relative to categorical approaches (Barlow, 1991; Bentall, Jackson, & Pilgrim, 1988; Blashfield, 1984; Meehl, 1986; Mirowsky & Ross, 1989). It has been proposed that, rather than choosing only one approach to description in psychopathology, the categories of disorders routinely could be complemented by dimensional profiles (Lorr, 1986; McReynolds, 1989). For example, a psychopathological severity

axis could be added to future diagnostic systems (Lobo, 1989). Dimensional and categorical systems of description do not have to be mutually exclusive, nor antagonistic (Millon, 1991).

As for operational measures of disorder severity, a number of measures have been developed to assess the relative presence of paranoid and nonparanoid symptomatology. For example, a discriminant function analysis was performed by Gordon and Gregson (1970) on the Symptom Sign Inventory (SSI; Foulds, 1965) in an attempt to determine which items best separated paranoid and nonparanoid schizophrenics. By multiplying the ten most discriminating dichotomous items by their discriminant function weights, the authors developed a dimension for classifying patients into these two categories: the Weighted Symptom Sign Inventory (WSSI). The WSSI has since been employed successfully in investigations of the differences between the two patient groups (e.g, Neufeld, 1977). Such measures as the WSSI or the Maine Paranoid Scale (Magaro et al., 1981) could be used to develop a continuum of paranoid-nonparanoid symptomatology to reflect the severity of disorder factor.

Severity of Symptoms. As briefly noted earlier, it is hypothesized that schizophrenic patients at the two ends of the above described continuum display different symptoms. In other words, the symptoms which are displayed by the patient are dependent on the severity of his or her disorder. As Figure 1 indicates, the severity of the displayed symptoms -- their frequency and protuberance -- is seen as independent of the type of displayed symptoms (i.e., the severity of the disorder). The symptom may not be at a severe level but may indicate a more severe form of disorder. By analogy, in the arena of physical illness, extreme

skin disfigurement may be an indicator of chronic superficial inflammation (lupus erythematosas discoid) while a less extensive disfigurement may herald a general connective tissue disorder with lethal abnormal immunologic sequelae (lupus erythematosus systemic; Friel, 1981).

The type of symptoms that would be part of such a model would include the various positive and negative symptoms discussed in the DSM-III-R (e.g., delusions, incoherence, blunted affect, social isolation). These symptoms could be measured by currently available symptom rating scales such as the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1984a) and the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984b). These two scales use a combination of the symptom's frequency, its strength, and the effect it has on the patient in determining ratings on a 6-point scale. Both general symptom ratings (e.g., global rating of severity of hallucinations) and more specific ratings (e.g., voices conversing, olfactory hallucinations) are available.

The symptoms of schizophrenia neither appear to be exclusive to the disorder nor relegated only to floridly pathological states. Emotional blunting, tangential speech, and delusions are present in a variety of psychopathologies (Newmark, Raft, Toomey, Hunter, & Mazzaglia, 1975). Moreover, such symptoms extend to nonpatient samples (e.g., Chapman & Chapman, 1980, 1988; Strauss, 1969) and may be related to such variables as culture and religion (Andrade, Srinath, & Andrade, 1988; Newhill, 1990; Spanos & Moretti, 1988; Westermeyer, 1988).

It has been recognized by many researchers that the symptoms of schizophrenia can be viewed as being on continua. In describing

schizophrenia, Bleuler (1911/1950) wrote "...it is extremely important to recognize the (symptoms) exist in varying degrees and shadings on the entire scale from pathological to normal" (p. 13). In more recent years, the positive symptoms, such as thought disorder (e.g., McConaghy, 1989) and delusions (e.g., Butler & Braff, 1991; Chadwick & Lowe, 1990), have been described as being on a continuum with normal behaviour. Discussing the negative symptoms of schizophrenia, Barnes (1989) wrote: "If they are considered as a loss of normal function then, by definition, severity must be rated on a continuum from normal" (p. 8) Research generally has supported this view (cf., Tandon & Gredon, 1991)

The extension to nonpatient samples can be illustrated in the case of hallucinations. The experience of brief or "benign" hallucinations among no..psychiatric patients has been reported for many years as occuring quite outside existing or future clinical levels of psychopathology (e.g., Forrer, 1960; Jansson, 1968; Mott, Small, & Anderson, 1965; Sedman, 1966). Further evidence indicates that hallucinations should not be viewed . rare experiences. For example, Posey and Losch (1983) surveyed 375 college students as to whether or not they nad ever experienced auditory hallucinations. The hallucinatory material ranged from "talking to dead relatives" to "hearing the doorbell or a phone ring when it didn't". Two hundred sixt·-eight of the students (71%) reported having at least one experience of at least one of these hallucinations. Posey and Losch concluded that such hallucinations are common experiences that usually are not reported. Thus, frequent hallucinations can be viewed as extensions of a "normal" human capacity for hallucinatory experience.

Several factors long have been identified as related to the experience of brief or "benign" hallucinations. For example, in 1883, Galton enumerated some conditions which he viewed as conducive to "visions" in his colleagues and relatives, such as solitary musing or a lack of sleep (see Slade, 1976). Early neurologists, such as Hughlings Jackson, recognized that physiologically abnormal states could result in the development of hallucinations from environmental stimuli (Sacks, 1970). Assad (1990) reviewed the more recent research and listed numerous factors seeming to bring about hallucinations in otherwise non-morbid states (e.g., grief, life-threatening stress, religious experiences, social deprivation). Physiological factors such as neurological disorders, eye and ear diseases, alcoholism, various forms of deprivation (sensory, sleep, food, and water), and drug side-effects have also been identified as etiological factors in episodes of hallucination (Assad, 1990).

Hallucinations can be defined as "positive" symptoms -- instances where "normal functions" have been replaced, or added to, by other functions (see, e.g., Chapman & Chapman, 1973). The current formulation requires that, in principle, symptom occurence is not all-or-none, but can take place with greater or lesser degrees of frequency and intrusiveness. As thorough a discussion of negative symptoms seems less urgent since they are defined as a deterioration of normal functioning (Barnes, 1991). Reduced functioning in normals is evident under numerous circumstances, such as during periods of exhaustion or periods of intense emotion. The loss, therefore, of function associated with negative symptoms once again can be viewed as ranging from relatively innocuous to extreme.

Describing the symptoms of schizophrenia as continuous in this way does not negate schizophrenia as a distinct clinical disorder. As Meehl and Golden (1982) have stated, such a conclusion "...is as bright as saying that there can't be such a thing as meningitis since one of its tests is high fever, and all degrees of fever exist in sick people!" (p. 136).

The symptom severity continuum may suggest additional avenues of inquiry into patient status. For example, the effects of antipsychotic medication may be viewed from a symptom-severity perspective. Medication most often "controls" symptomatology, reducing its severity, but seldom removing the disorder or its symptoms in entirety. It is a common clinical observation that under medication a patient may experience symptoms to some degree (e.g., auditory hallucinations) but be able to cope with them more successfully. For example, Kay and Lindenmayer (1991) have found that when chronic schizophrenic patients were treated with clozapine, the severity of their symptoms lessened. Nevertheless, the intercorrelations among symptoms remained stable, indicating that the symptom pattern was consistent even when the symptom severity level was pharmacologically controlled. Observe as well the common re-emergence of symptom severity among patients who discontinue medication. Likewise, those individuals displaying the "negative" symptoms consistent with the suggested more severe form of disorder (e.g., nonparanoid symptoms such as affective blunting) may be more refractory to otherwise therapeutic dosages of medication (Crow, 1980a).

Aims of the Present Investigation

The primary aim of this investigation was to determine the nature of the relation between paranoid and nonparanoid schizophrenia. It was

hypothesized that the relation would approximate the above described reconceptualization. That is, paranoid and nonparanoid schizophrenia could be seen on a continuum similar to that outlined for the present model's severity of disorder factor. In order to investigate this hypothesis, the following study was undertaken (See Table 3). One hundred patients with a diagnosis of either paranoid schizophrenia or nonparanoid schizophrenia were rated on a wide variety of different assessment devices of paranoid and nonparanoid schizophrenic symptomatology. The groups were also compared on several "control" scales; that is, scales which did not reflect symptomatology differences between those patients with paranoid schizophrenia and those with nonparanoid schizophrenia.

The schizophrenia assessment devices were developed by several methods. For example, some of the measures were based on results from factor analysis. Other measures had been developed from an a priori theoretical base. Some were measures of the subtype itself; either overall paranoid symptomatology or overall nonparanoid symptomatology. All of these assessment devices, however, were designed specifically for schizophrenia and all had reported adequate levels of reliability and validity.

The primary aim of the present study was to determine if the paranoid/nonparanoid distinction is best viewed as continuous or discrete. Two recently developed methods were employed to examine this question: maximum covariance analysis (Meehl & Golden, 1982) and comparsions of base rates estimates (Gangestad & Snyder, 1985). These methods have both been employed in previous investigations to determine

Table 3

Outline of Study Design
(Page 1 of 2)

STEP 1

Collect data on 100 subjects (both inpatients and outpatients) with a diagnosis of schizophrenia at an Ontario Psychiatric Hospital. Data will include:

Demographic/history:
- age
- estimate of age of onset
- hospital casebook number
- current patient diagnosis
- current hospitalization status
- history of street drug use/abuse
- current medications

Symptom ratings based on structured clinical interview/chart notes:
- Symptom Rating Scale
- Brief Psychiatric Rating System
- Symptom-Sign Inventory (43 questions)
- Maine Paranoid Scale
- Schedule for the Assessment of Positive Symptoms
- Schedule for the Assessment of Negative Symptoms

WAIS-Clarke IQ estimate

Sternberg Choice Reaction Time Task

Table 3

Outline of Study Design
(Page 2 of 2)

STEP 2

Perform a set of preliminary scale-level data analyses to review data and determine its adequacy for further analyses.

Descriptive statistics

Reliabilities

Correlations

Comparisons between groups:
- pre-analysis based (e.g., gender, acute/chronic)
- data based (e.g., Maine Paranoid Scale paranoid/nonparanoid)

STEP 3

Perform a series of item-level analyses to investigate the dimensional vs categorical structure of the data. The schizophrenia and control data analyses were performed separately.

Item reliabilites

"Judicious Pruning" of the two data sets

Cluster analysis to determine symptom clusters

Rate nature of schizophrenia data clusters along two parameters:
- Paranoid / Nonparanoid / Both/Neither
- Positive / Negative / Both/Neither

A series of three iterative consistency tests for each of five symptom cluster groups

Maximum covariance analyses for the cluster groups

Two base rate estimates for the cluster groups

Comparison of the base rate estimates

if dimensional models or categorical models are more appropriate to describe data sets.

Maximum Covariance Analysis. Meehl and Golden (1982) developed Maximum Covariance Analysis (MAXCOV) as a method of detecting if an underlying latent class exists in data which do not have any perfect external criteria. Gangestad and Snyder (1985) described MAXCOV as follows:

> If a class variable underlies the responses to the items as conjectured, if the classes are of not too unequal size ... then the seven sample covariances between (the two other scores) as a function of the values on the 7-point scale associated with the samples should be peaked, maximal toward the middle values and nearer to the zero toward the extremes. If no class variable exists, no reason exists to expect a peaked covariance curve. (p. 326, italics in original)

That is, if two classes do exist in a variable group, then the covariance between two symptom clusters would be a function of the relative proportions of the two classes (i.e., taxon and non-taxon; see Figure 3). If there were no class variable, then the covariance would always be close to zero as there would be no peak in the curve (see Figure 4) No statistics have yet been devised to determine if a "peak" actually exists. Instead, "eye-balling" the resultant curve seems to be all that is available at present (Golden, 1991).

Gangestad and Snyder examined the validity of this technique by comparing the curves generated by two personality variables (see Figure 5). One variable, self-monitoring was hypothesized to be made of discrete classes. The other variable, impulsivity, was hypothesized as

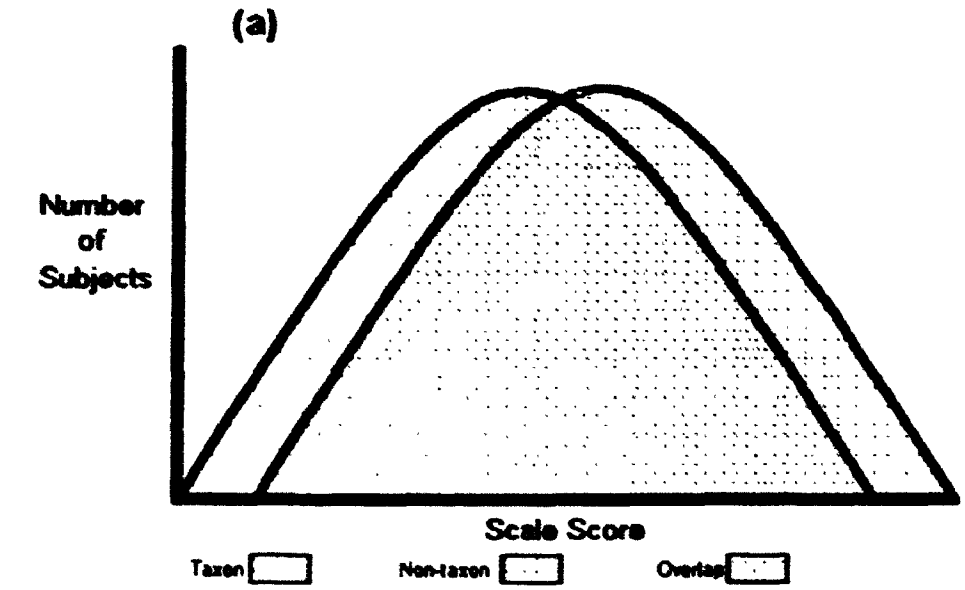
FIGURE 3

MAXCOV Example:

(a) overlapping distributions

of equal-sized taxon and nontaxon

(b) resultant covariance curve


(a)
Number of Subjects
Scale Score
Taxon
Non-taxon
Overlap

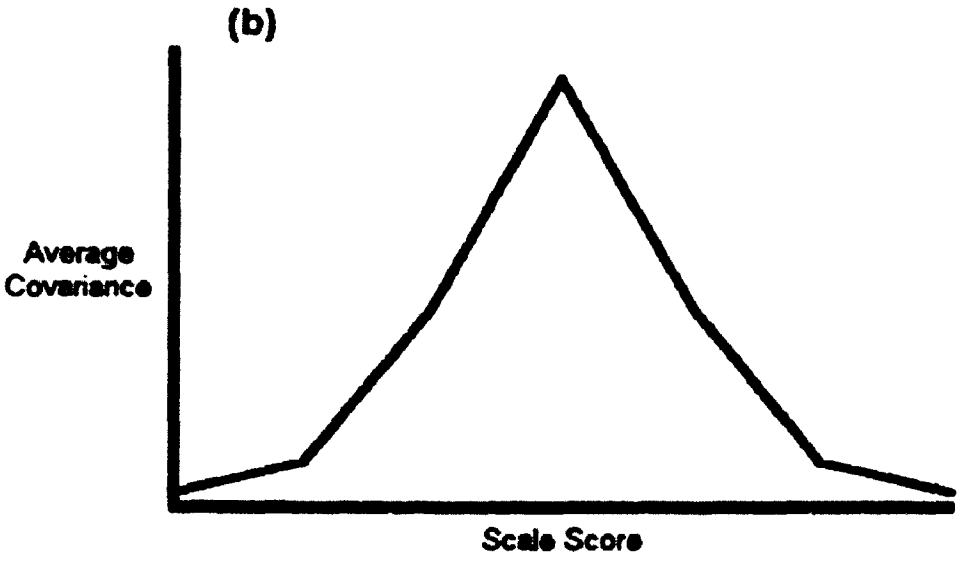
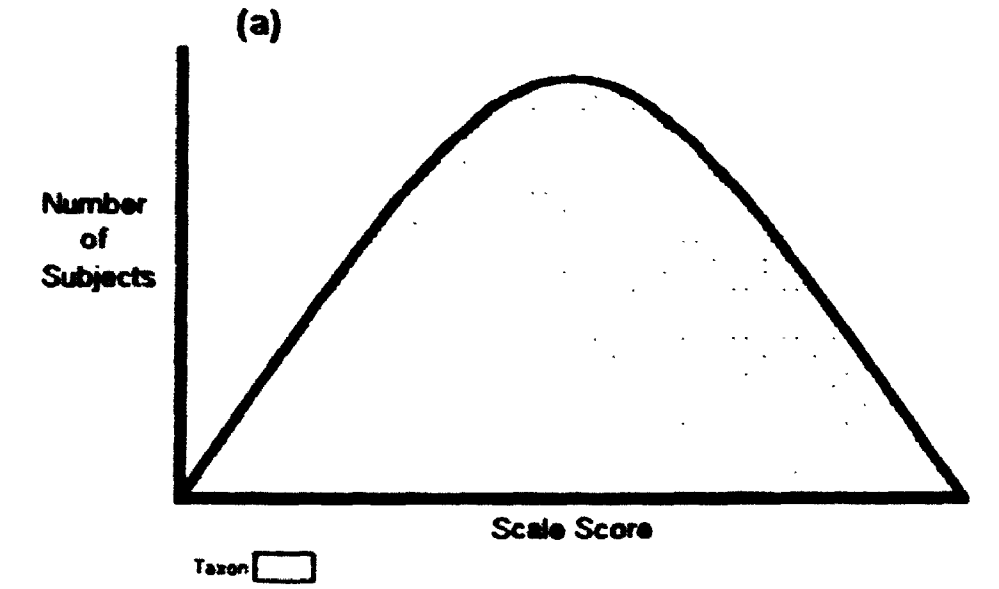

(b)
Average Covariance
Scale Score

FIGURE 4

MAXCOV Example:

(a) distribution of single nontaxonic sample

(b) resultant covariance curve


(a)
Number of Subjects
Scale Score
Taxon

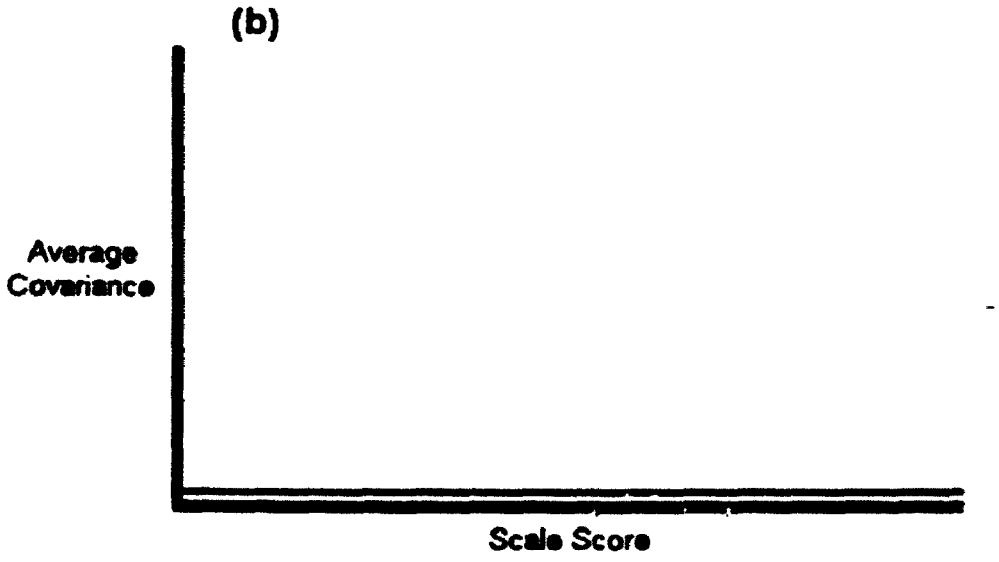
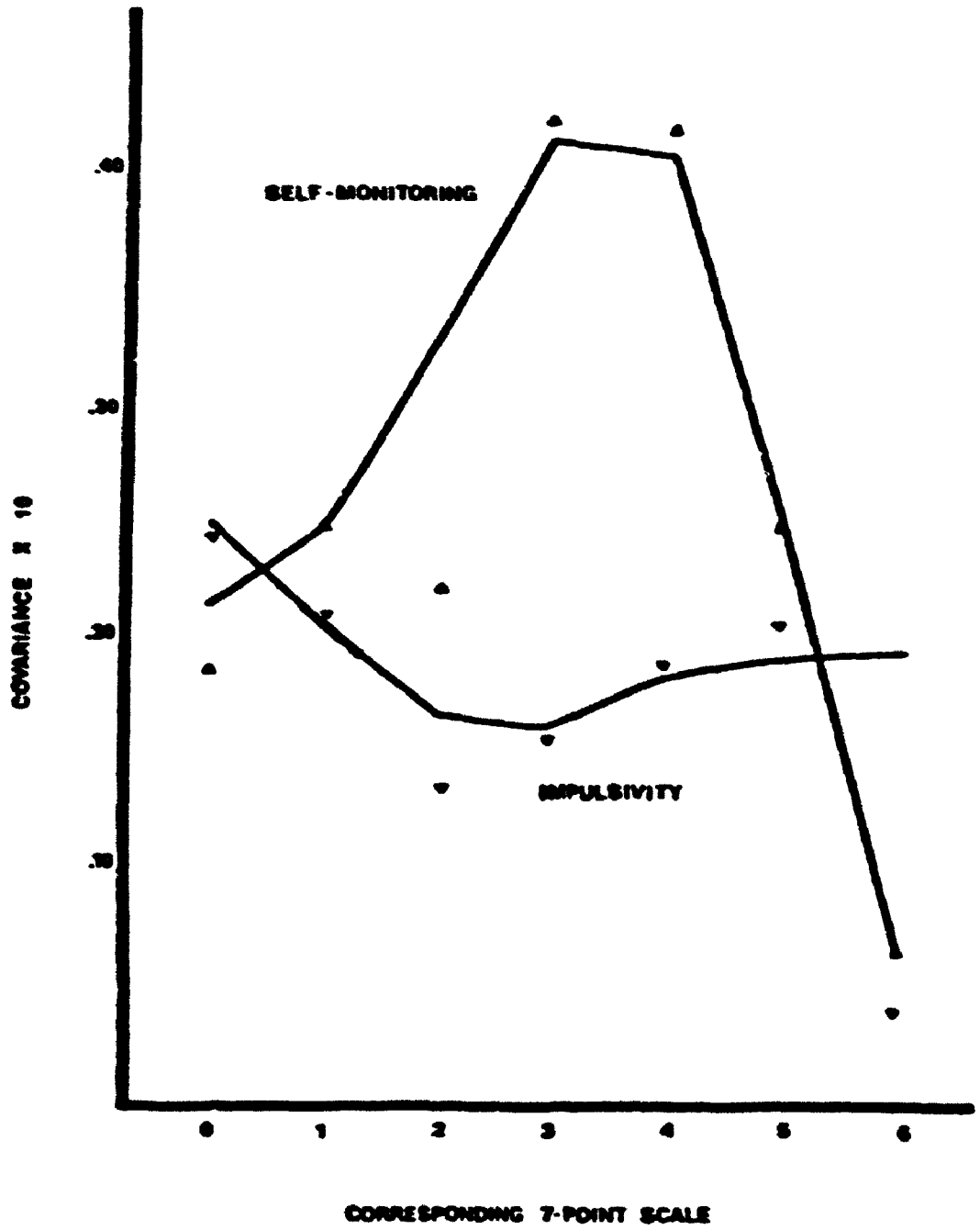

(b)
Average Covariance
Scale Score

FIGURE 5

Covariance between best self-monitoring items and best impulsivity items as a function of corresponding 7-point scales. (Solid lines represent smoothed functions. Upright triangles represent raw self-monitoring data points. Inverted triangles represent raw impulsivity data points.)

(p. 329; Gangestad & Snyder, 1985; reprinted with permission)


SELF-MONITORING
IMPULSIVITY
COVARIANCE X 10
0
1
2
3
4
5
6
CORRESPONDING 7-POINT SCALE

dimensional. Results indicated that self-monitoring followed the curve predicted by Gangestad and Snyder (i.e., peaked in the centre). In comparison, impulsivity failed to display such peakedness. Thus, they concluded that the maximum covariance analysis appropriately detected a latent class structure.

Trull et al. (1990) also supported MAXCOV by comparing two different curves. One curve was based on the MAXCOV analysis of items reflecting borderline personality disorder and was thought by Trull et al. to be dimensional. Therefore, they predicted a flat line. An examination of Figure 6, however, indicates that it has an upward-curving right-hand peak of approximately 0.25. Subsequent MAXCOV research has indicated that similar peaks are indicative of the presence of a taxon/class with a base rate of less than 25% (see below). More important for the present discussion, however, is the second curve which was based on items reflecting biological gender. Trull et al. assumed that the MAXCOV analysis would result in a peaked curve for gender because of the presence of two classes. This final prediction were borne out by the results of their analyses. The smoothed curve ranged from approximately 0.00 at the ends to a central peak of approximately 0.25.

As noted above, some MAXCOV analyses have resulted in an upward rise at the right end of the resultant curve. Recent Monte Carlo studies have found this pattern to be related to a class model when the base rate for one of the classes in the sample was less than 0.25 (Meehl, personal communication, October 28, 1992; see Figure 7). Results supporting this interpretation of a curve's upward-curving right-hand peak was found by Lenzenweger and Korfine (1992) in a MAXCOV

FIGURE 6

Mean covariance of 28 possible pairs formed from eight borderline (n = 409) and eight male sex (n = 244) items as a function of corresponding 7-point scales (p. 45; Trull, Widiger, & Guthrie, 1990; reprinted with permission)

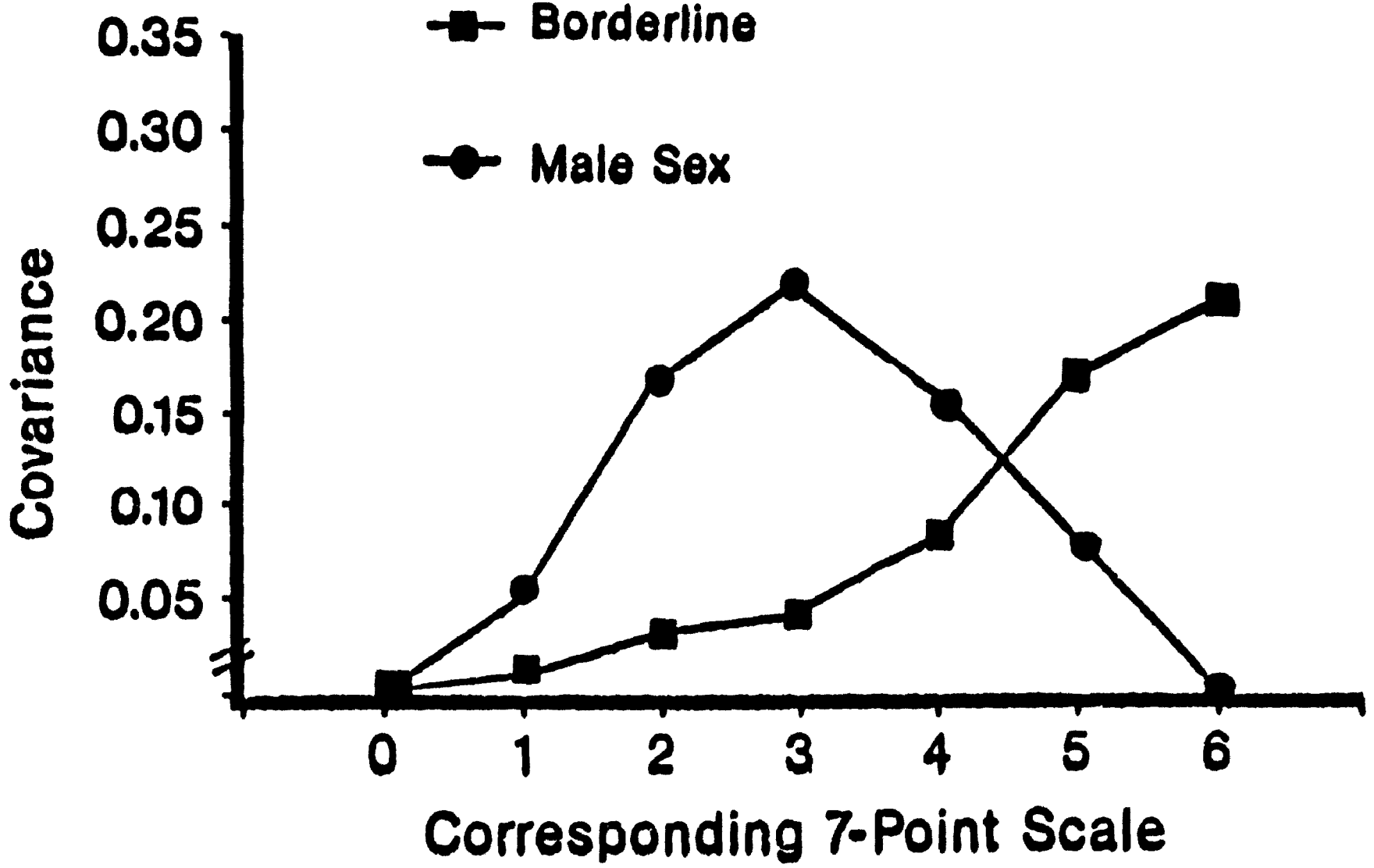

Borderline
Male Sex
0.35
0.30
0.25
0.20
0.15
0.10
0.05
Covariance
0
1
2
3
4
5
6
Corresponding 7-Point Scale

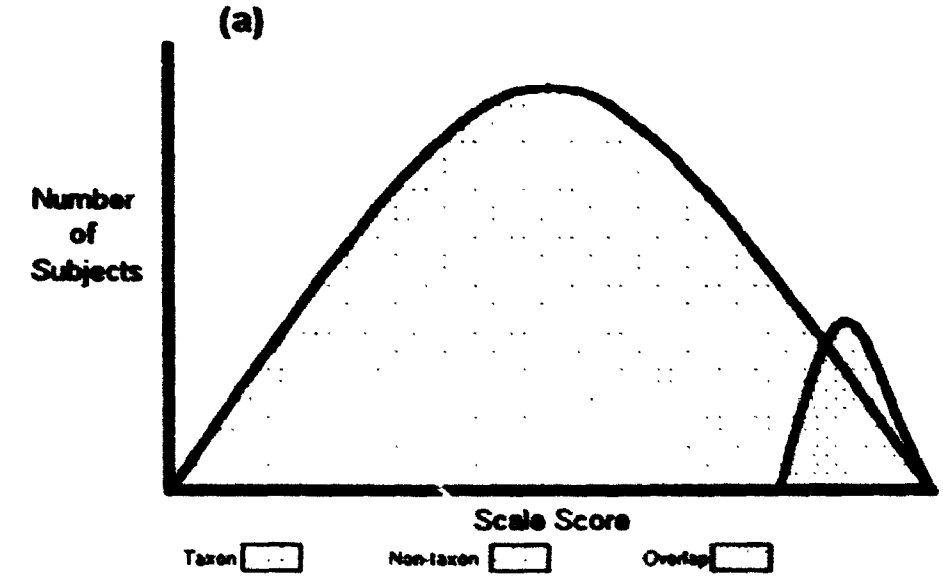
FIGURE 7

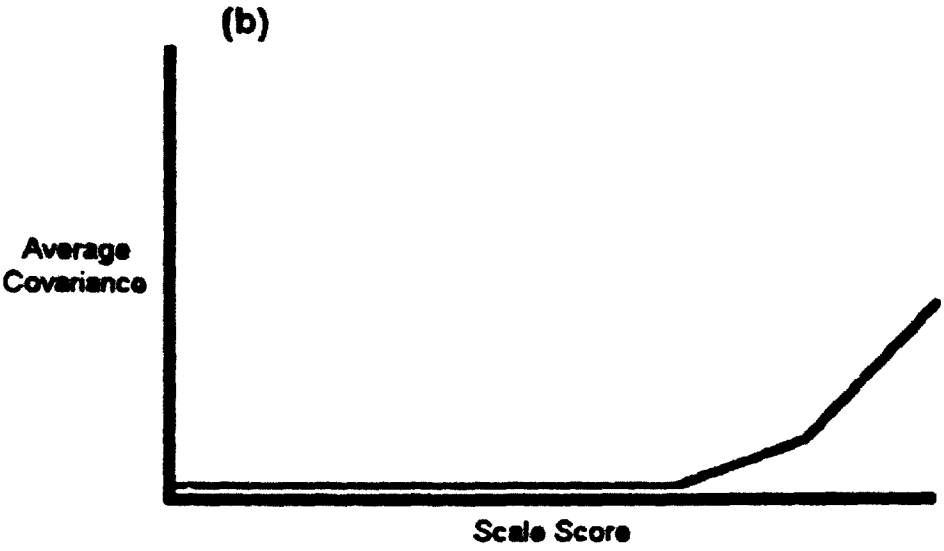
MAXCOV Example:

(a) overlapping distributions

of unequal-sized taxon and nontaxon

(b) resultant covariance curve


(a)
Number of Subjects
Scale Score
Taxon
Non-taxon
Overlap


(b)
Average Covariance
Scale Score

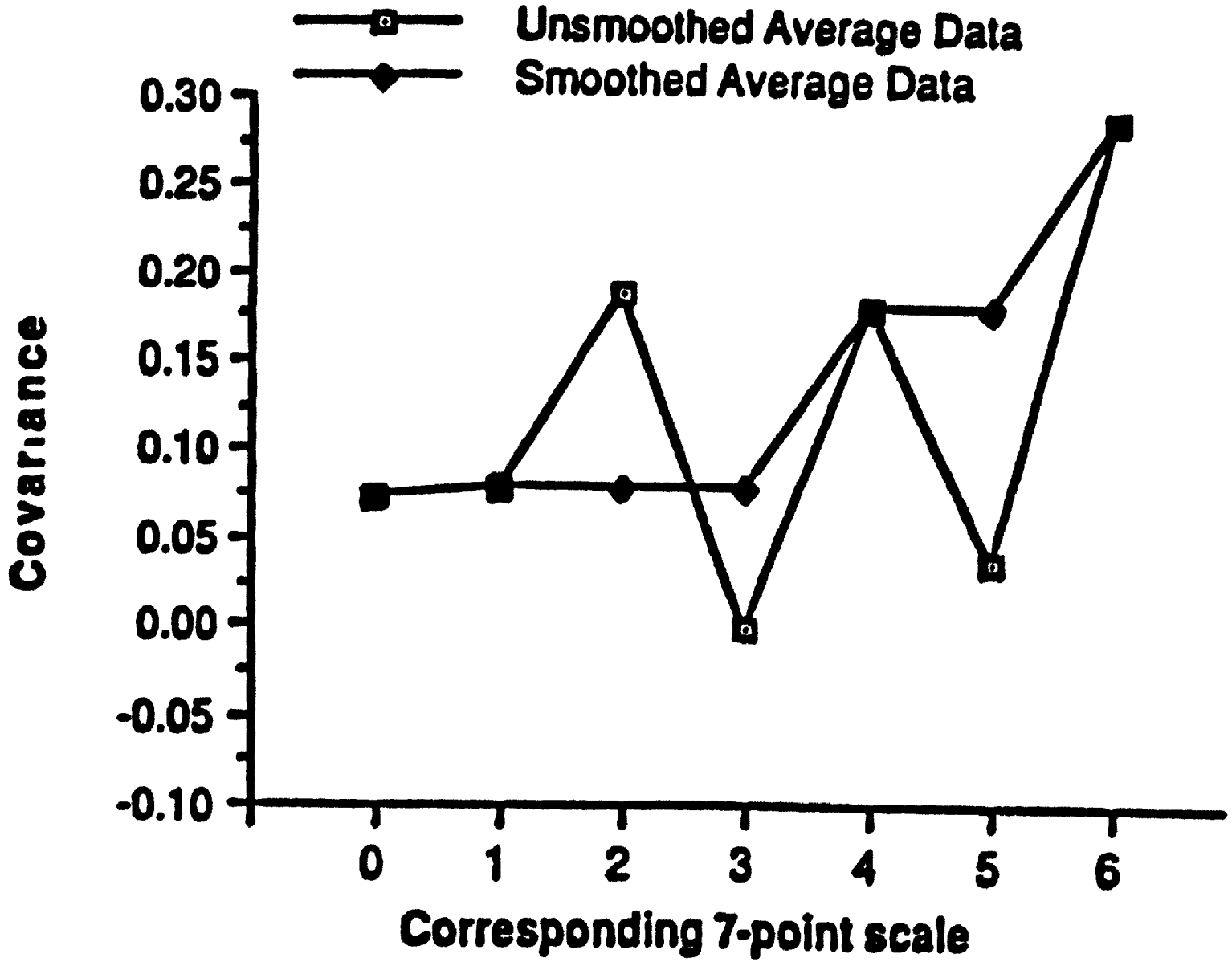
analysis on schizotypy in a university undergraduate program (see Figure 8). In that instance, the resultant smoothed curve ranged from approximately 0.07 to a right-hand peak of approximately 0.30.

Trull et al. (1990) reviewed six methods proposed for the empirical determination of whether a construct is categorical or dimensional. The methods they reviewed included factor analysis, cluster analysis, bimodality/multimodality, admixture analysis, discontinuous regression against an external variable, and MAXCOV. They reported technical problems with many of these techniques and referred to a number of cogent technical reviews to support their points (e.g, Grayson, 1986, 1987). Also, factor analysis and cluster analysis were seen as inadequate since they had an a priori theoretical preference for one model over the other. For example, cluster analysis will create subgroups regardless of whether or not they actually exist. Other methods, such as bimodality/multimodality, admixture analysis, and discontinuous regression, all require an accepted external criterion. Because their study of borderline personality disorder did not have this type of "gold standard" for the ends of their proposed continuums, Trull et al. employed MAXCOV. Since the present investigation also did not have external "gold standards" for defining the ends of proposed continua, MAXCOV was employed for these analyses.

MAXCOV has been successfully employed in the investigations of biological sex detection (Golden & Meehl, 1980), dementia (Golden, 1982), self-monitoring (Gangestad & Snyder, 1985), tardive dyskinesia (Golden, Campbell, & Perry, 1987), depressive syndromes (Grove et al., 1987), neonatal brain dysfunction (Golden, Vaughan, Kurtzberg, & McCarton, 1988), Type A personality (Strube, 1989), borderline

FIGURE 8

Covariance curves based on the average covariances across all 28 pairings for each interval on the corresponding 7-point scale for both unsmoothed and smoothed data (N = 1,093) (p. 570; Lenzenweger & Korfine, 1992; reprinted with permission)


Unsmoothed Average Data
Smoothed Average Data
Covariance
0.30
0.25
0.20
0.15
0.10
0.05
0.00
-0.05
-0.10
0
1
2
3
4
5
6
Corresponding 7-point scale

personality disorder (Trull et al., 1990), and schizotypy (Lenzenweger & Korfine, 1992). The use of this technique has also been suggested for future research in personality disorders (Grove & Tellegen, 1991), melancholia (Widiger & Trull, 1991), as well as schizophrenia (Tsuang, Lyons, & Faraone, 1990).

Comparison of base rate estimates. The primary hypothesis underlying this method was described by Gangestad and Snyder (1985) as follows:

> If a latent dichotomous class variable does underlie the responses to the indicators, then a specific parameter - the proportion of the population belonging to one class and the proportion belonging to the other class - defines the distribution of the class variable (p. 326).

In other words, "if different methods to estimate the base rates fail to provide consistent estimates, either the class model (underlying the estimates) was inappropriate or some auxilliary assumptions of the methods have not been satisfied" (p. 327). Thus, different sets of mathematical formulae for estimating base rates, which were not derivable from one another, should provide consistent results only if there was some form of an underlying dichotomy.

The present investigation employed two of the three base rate estimation techniques adopted successfully by Gangestad and Snyder. These two methods were outlined by Meehl (1973) and Golden and Meehl (1979). The third method, described by Golden (1982), was exceedingly complex and beyond the scope of the present investigation without customized computer programs. Gangestad and Snyder found that the three methods yielded "remarkably consistent estimates" (p. 327) for the

personality variable (self-monitoring) they believed was discontinuous and they found to have a peaked MAXCOV curve. When these base rate estimate techniques were applied to a personality variable (impulsivity) which was viewed as continuous and which had a flat MAXCOV curve, the resultant base rates were not consistent with one another. Thus, Gangestad and Snyder concluded that a comparison of these three base rate estimate techniques could be employed as a measure of classes versus dimensionality by examining the uniformity of their results.

If the results of the MAXCOV and base rate comparisons were consistent with a dimensional model, then initial research support would be available for the severity of disorder factor of the proposed two-factor model of the symptomatology of schizophrenia. It would also support a Bleulerian, and not Kraepelian, conceptualization of schizophrenia. That is, schizophrenia would be seen as a single disorder with different manifestations and not as different disorders with commonalities of symptomatology. Such results would have a number of significant implications for research (e.g., single gene locus studies) as well as clinical practice (e.g., diagnostics).

CHAPTER 2 - METHOD

Subjects

The subjects were 100 patients, tested between December 1988 and August 1990 at London Psychiatric Hospital (LPH). A sample size of 100 is thought to be the minimum number of subjects required for the Maximum Covariance analysis (Golden & Meehl, 1979). While larger sample sizes are advised, preliminary Monte Carlo results indicate that the presence of a taxon may be detected with a sample size as low as 100. These results suggest that while variability increases, there is no concomitant interference with the resultant taxonic curve shape (Meehl, personal communication, October 28, 1992).

The patients in this study had been diagnosed by psychiatrists at LPH as having schizophrenia. It was noted whether a patient's diagnosis was for the paranoid type (n = 45) or not (n = 55). The study did not include patients who were diagnosed as having a schizoaffective disorder. This step was taken because research indicates that the degree to which such a patient has schizophrenia would be in doubt (Yasamy, 1987).

No psychiatric control group was used in the present study. The primary aim of this research was to investigate the nature of schizophrenic symptomatology. As such, no specific relations were proposed involving other psychiatric groups. Also, some of the negative symptoms of schizophrenia are strongly related to other psychiatric disorders (e.g., social withdrawal in a major depressive disorder) while other symptoms are not as strongly related (e.g., catatonic posturing). Likewise, many of the control scales specifically relate to other psychiatric disorders (e.g., BPRS "Anxious Depression" factor). Adding

a psychiatric control group which has this form of uneven interrelation with the scales was outside the scope of the present investigation.

Several variables initially were investigated to ensure that the present data were consistent with previous research on schizophrenia. Some of these variables included patient demographic and history data and will be described in this section. In addition, the scale-level symptom ratings, reviewed briefly in the following section on Paranoid/Nonparanoid Symptom Measures, were similarly analyzed. The results of these analyses are secondary, however, to the primary MAXCOV and base rate comparison analyses. A brief overview of these preliminary results are included at the beginning of the next chapter. A more thorough description of these analyses is contained in Appendix U.

To investigate the effect of chronicity, the patients were divided into acute (n = 31) and chronic (n = 69) subgroups. The acute patients had their initial diagnosis at LPH within the previous three years (inclusive). The patients who had their initial diagnosis at LPH more than three years ago were classified as chronic. Previous research indicates that after the first three years, further changes in displayed symptomatology are significantly lessened (Huber, Gross, & Schutteer, 1975; Strauss, 1973). It is realized that, as with the initial diagnosis of the schizophrenia criterion, this distinction may not be reliable. Nevertheless, it does offer a crude benchmark for some exploratory analyses of change of symptoms over time.

These results were also compared to the hospital casebook number. These numbers are assigned consequetively to new patients registered at LPH. It is expected that there would be a significant relationship

between length since initial hospitalization and this casebook number. Therefore, this number was included as another broad estimate of chronicity.

All of the patients also met the following series of additional criteria: fluent in English, age between 18 and 65, at least a Grade 8 education, an IQ of at least 80 (as assessed by the WAIS-Clarke IQ score), no documented evidence of organicity, no evidence of epilepsy, and no history of electroconvulsive therapy (ECT) within the previous six months. It should be noted that seven patients did not complete the WAIS-Clarke IQ test. Three patients did not return for the second session, which included the WAIS-Clarke. Three other patients did not have their reading glasses and were unable to read the test. The seventh patient was too psychotic by the time of the second session to understand the test's instructions. All seven of these patients had completed successfully some high school education and there was no evidence in the chart of concerns regarding possible intellectual impairment. It was therefore decided to include these seven patients.

It should be noted that no exclusions were made as a result of a history of drug or alcohol use. This position was based on three points. First, drug and alcohol use is common among schizophrenic patients (Dixon, Haas, Weiden, Sweeney, & Frances, 1990; Drake, Osher, & Wallach, 1989). Second, patients with schizophrenia who also have a history of substance abuse have not been found to be more impaired or more symptomatic when compared to patients who have been lifelong abstainers (Zisook et al., 1992). Third, a DSM-III-R "diagnosis (of schizophrenia) is only made when it cannot be established that an organic factor initiated and maintained the disturbance" (American

Psychiatric Association, 1987; p. 192). Such organic factors include "Organic Mental Disorders" which have symptoms which might suggest schizophrenia (e.g., "Alcohol Hallucinosis", "Amphetamine or Similarly Acting Sympathomimetic Delusional Disorder"). As a consequence of these three points, it was decided that excluding patients who use alcohol or drugs (or both) would unnaturally bias any sample of schizophrenics which the study employed. Nevertheless, in an effort to determine how drug use might relate to symptoms, it was noted which patients had a history of substance use or abuse (n = 65).

It should also be noted that the gender ratio in the present investigation was not equal (Male:Female (M/F) ratio = 69:31 = 2.22). The DSM-III-R description of schizophrenia states that the "disorder is apparently equally common in both sexes" (p. 192). Yet, when the initial DSM-III field trial data is analyzed for gender differences in diagnosis, this statement is not supported (Williams & Spitzer, 1983). Of the 20 Axis I diagnostic categories with at least 10 males or females, schizophrenia had the fourth highest M/F ratio (258:170 = 1.52). Higher M/F ratios have since been reported in other research on the DSM-III diagnosis of schizophrenia (Lewine, Burbach, & Meltzer, 1984; M/F = 34:12 = 2.83). In reviewing the research on such differences, Goldstein, Santangelo, Simpson, and Tsuang (1990) concluded that, while schizophrenia may be present in both genders, its prevalence may be different. Thus, the unevenness in gender representation is not considered to be unique to this study.

Five other patient variables were assessed to determine if there were significant differences among the groups: age at time of participation in study, age at time of initial hospitalization at LPH,

IQ scores, medication dosage and study subject number. IQ was assessed by the WAIS-Clarke multiple-choice version of the WAIS vocabulary test (Paitich & Crawford, 1970). This short form has been used frequently (e.g., Alexitch, Blackstein, & Flett, 1988; Giles & Shaw, 1987; Haaga, DeRubeis, Stewart, & Beck, 1991; Naranjo, Kadlec, Sanhueza, Woodley-Remus, & Sellers, 1991; Wilkinson, Leigh, Cordingley, Martin, & Lei, 1987) and has been shown to correlate with the results of a full scale IQ meausure. It should be noted that the WAIS-Clarke IQ test meets the three criteria for employing a short-form IQ test as put forth by Silverstein (1990) in his review of the topic; that is, (1) a high or highly significant correlation with the original scale, (2) no significant difference in mean IQ when compared to the original scale, and (3) a low level of disagreement with the original scale in classifying subjects. Its use in this study is also in accordance with one of Silverstein's categories for the legitimate use of such a short form in research. After reviewing the literature on the topic, he wrote that it can be legitimate in situations where "...all that is required is a quick check on an individual's intellectual status (e.g., when the assessment of an individual's intelligence is peripheral to the reason for referral)" (p. 9).

In the present study, the mean WAIS-Clarke IQ scores was 98.7 (σ = 8.36) and ranged from 80 to 115. The 44 patients with a current hospital diagnosis of paranoid schizophrenia averaged 99.4 (σ = 8.37) with a range of scores from 80 to 115. In comparison, the patients with nonparanoid schizophrenia had a mean score on the WAIS-Clarke of 98.2 (σ = 8.4) and also ranged from 80 to 115. An independent groups t-test

showed no significant differences between the two groups types of patients ($t(91) = 0.73$, ns).

Medication dosage was taken from current hospital records. Medication dosage was examined by converting medication histories to chloropromazine (CPZ) equivalency units (Davis, 1976; Lehman, 1975; Wyatt & Torgow, 1976). In other words, the level of medication is translated to a level of CPZ which would result in an equivalent degree of therapeutic effectiveness (e.g., 5 mg of Trifluoperazine (e.g., Stelazine) = 100 mg of CPZ (e.g., Largactil, Thorazine)). This method is recommended as the method for describing the mean neuroleptic dose across a range of patients with schizophrenia (Blanchard & Neale, 1992b).

The ratios for these transformations were chosen from a table of neuroleptic doses in the Clinical handbook of psychotropic drugs (3rd ed. - rev.) written by a team of pharmacologists and psychiatrists at the Clarke Institute of Psychiatry (BezchlibnyK-Butler et al., 1992). Since this listing is more recent than other listings and, as a result, may not yet be familiar, the reasons for the choice of it over older listings will be reviewed. First, this version's CPZ equivalency units both significantly correlate with, and do not significantly differ from, those equivalencies reported by Lehman (1975; $r(9) = 0.98$, $p < 0.001$; $t(10) = 0.50$, ns), by Davis (1976; $r(9) = 0.98$, $p < 0.001$; $t(10) = 0.90$, ns), and by Wyatt and Torgow (1976; $r(5) = 0.95$, $p < 0.001$; $t(6) = 0.68$, ns). These previous listings have been employed several times in previous research (e.g., Morrison et al., 1990). Second, a wider number of psychotropic medications are included on the BezchlibnyK-Butler et al. list ($n = 24$) than are on those lists developed by Lehman, Davis,

or Wyatt and Torgow (n = 18, n = 19, and n = 11 respectively). Third, this larger list includes recently developed psychotropic medication (e.g., Pimozide (e.g., Orap)). When these factors are considered, the choice of this list over previous lists can be supported.

The investigation of medication is important because it is theorized that it affects symptom levels but it does not reduce the severity of the actual disorder (Nicholson & Neufeld, in press). Antipsychotic medications, such as CPZ, usually are administered to a level of clinical efficacy. That is, the amount administered reflects the amount necessary to eliminate (as much as possible) some of the symptoms. Thus, this dosage differs among individuals and should not correlate with the level of residual symptomatology. As a result, the schizophrenic symptomatology scales will only measure the residual level of symptom severity (Neufeld & Broga, 1981). It is believed that the severity of the disorder factor will indicate itself in a clear fashion even if the severity of symptoms factor reaches an imposed "ceiling" effect due to the efficacy of the medication. Also, while the use of the CPZ equivalency units may be inadequate to control for the effects of medication, it does allow for inferences to be made regarding the clinical status of the subjects (Blanchard & Neale, 1992a).

It was not expected that there would be differences in the level of medications between the patients with paranoid schizophrenia and those patients with nonparanoid schizophrenia. Nor was a differential group effect expected in *how* these medications would affect the results of the investigation. Nonetheless, it was believed safest to measure for any significant discrepancies which might occur. Statistical control of these discrepancies (e.g., analysis of covariance) is generally

contraindicated (Neufeld, 1977). Actual group differences may be removed inadvertantly by such a procedure when these differences are not distinguishable statistically from group medication differences (Cochran, 1957). As a result, any significant patient group differences in medication which might have existed were considered when the results of the present research were interpreted.

Finally, the subject's study number (001 through to 100) was included as a control variable. Significant differences between groups of patients or significant correlations with other variables would give valuable process information about the study. For example, if a measure of volition was positively correlated with subject number, then we would need to determine if the latter patients actually had more volition or if the investigators were reporting it more often. It was therefore included in preliminary analyses involving demographic data.

Subject Recruitment

All patients in the present study were either inpatients (n = 70) or outpatients (n = 30) from London Psychiatric Hospital. To ensure that the investigators were blind to the history and diagnostic subtype for each patient as well as to ensure that the patient's rights were not violated, a series of steps were taken before any patient was tested. For inpatients, a research assistant (AMW) regularly reviewed the listing of current patients at LPH. If there were any patients who were diagnosed as schizophrenic (who were not already part of the study), AMW then asked for their previous hospital charts to be sent up from the LPH archives. One or two working days later, she reviewed these charts to investigate the criteria for the study. If the patient passed this hurdle, AMW would go to the patient's ward and review the current chart

with regard to the above-described criteria. One hundred ninety-eight patients passed this stage of the review.

The primary investigator (IRN) was then given a list of suitable patients with the patient's name, hospital casebook number, hospital ward, and current hospital psychiatrist. A memo was then sent to the psychiatrist which asked for his or her permission to approach the patient to take part in the study. Permission was not given for 22 of the 198 patients. The reasons for refusal, when given, were varied and included "would interfere with ongoing treatment", "too anxious", "too violent", "troublemaker", "too paranoid", "can't stand any frustration", etc.. Forty-five other patients left the hospital under a variety of circumstances (e.g., discharged, absent without leave, failed to return from a pass, went to jail, signed self out against physician's advice) before they were approved or before they could be approached to take part in this study. Of the remaining 131 patients who were asked to take part in the study, after having the study briefly described to them, 70 agreed to participate. Thus, 53% of the inpatients approached, and 35% of the inpatients who initially met the criteria successfully were recruited for this study.

The three methods of approaching outpatients for the study were somewhat different than that used for inpatient recruitment. The first method involved approaching outpatients through the Outpatients Department at LPH. In March 1989, a list of LPH outpatients who went to the hospital's Active Treatment Clinic (ATC) was prepared by the clinic's chief psychiatrist. This list contained 139 patients who met the diagnostic criteria for our study. Memos were then sent to the patients' psychiatrists with lists of their appropriate ATC patients to

determine if it was permissable to approach these patients to be a part of the study. Permission was not given for 5 of these outpatients. AMW then reviewed the charts to ensure that the remaining 134 patients were suitable for the study. Those patients who met the remainder of the study's criteria were then checked against the ATC listing to locate their phone numbers. Those patients who had valid phone numbers were contacted by phone and approached to participate. Twenty-one of the outpatient subjects approached in this manner participated in the current investigation.

The second method of approaching outpatients involved following up on discharged inpatients. As noted above, 45 suitable inpatients left hospital before they could be approached to take part in the study. The current ATC listing was then reviewed to see if they were being seen as outpatients. Thirteen patients who were being seen and who had current phone numbers were recorded. Their psychiatrists were then approached to obtain permission to approach them as outpatients. All of these patients were approved and subsequently were approached to take part in the study. Six patients in the study were recruited in this manner.

The third method of recruiting outpatients for the study involved a second separate list from the ATC chief psychiatrist obtained in June 1990. This list of 13 patients were under this psychiatrist's care and no further consent was necessary in approaching them to participate in the study. One patient had been seen earlier as an inpatient, leaving 12 for AMW to check. Two patients did not meet the criteria for the study and four patients had no current, valid phone listing in the ATC. Of the six patients contacted, three refused to participate. Thus, of the original list of 12 new patients from the ATC chief

psychiatrist, three patients became subjects in the present investigation.

Paranoid-Nonparanoid Symptom Measures

Symptom Rating Scale. In order to allow clinicians and researchers to measure psychiatric symptoms more reliably, Jenkins, Stauffacher and Hester (1959) developed the Symptom Rating Scale (SRS; see Appendix H). They reviewed previous research to outline the symptoms which had been noted as being important in clinical diagnosis. A series of studies and professional workshops were undertaken to outline the most reliable set of nonoverlapping symptoms. The final version of the SRS contained 20 symptoms which could be measured quickly and easily during the course of a standard 40- to 50-minute psychiatric interview.

Cohen, Gurel, and Stumpf (1966) administered the SRS 13 times over a four-year period to a sample of 1274 chronic schizophrenic patients in 12 hospitals. Results of their subsequent factor analysis indicated that there were five symptom factors present. (1) "Uncooperativeness" represented by a lack of cooperation during the interview. This pattern of symptoms was consistent although no clear reason for lack of cooperation was present. (2) "Depression-Anxiety" were consistently linked together in the factor analyses. (3) "Paranoid Hostility" is interpreted as characterized centrally by pathological suspicion and hostility, which Cohen et al. described as "an active paranoid schizophrenic response to interpersonal situations" (p. 43). (4) "Deteriorated Thinking" was defined as reflecting a qualitative deterioration indicative of schizophrenic thought pathology. (5) "Unmotivated" represented the pattern typically viewed as representative of "burned-out" chronic schizophrenics who are lacking in

goals, drive and ambition. Due to the large sample size studied over a long period of time across numercus settings, Cohen et al. argued that the results indicated that these five symptom factors were robust and were measured reliably by the SRS.

In the present investigation, three of these symptom factors (3, 4 and 5) were employed as measures of paranoid and nonparanoid schizophrenia (see Table 4). The third factor of the SRS, "Paranoid Hostility", was used as a measure of paranoid schizophrenic symptomatology. The fourth and fifth factors, "Deteriorated Thinking" and "Unmotivated" respectively, were used as measures of nonparanoid symptomatology. Two of these symptom factors, "Paranoid Hostility" and "Deteriorated Thinking", have been adopted in previous research as measures of paranoid and nonparanoid symptomatology (Magaro et al., 1981).

Previous research has been unclear as to how to develop the symptom scores from Cohen et al.'s results. In order to develop a subject's score for one of these factors, his or her scores on each of the 20 items were multiplied by the item's factor loading and the resultant scores were then totalled. (The factor loadings for the 20 symptoms are listed in Table 5.)

For the analyses which require single items, only the ten items which have factor loadings of 0.40 or greater were considered (items 5, 6, 7, 9, 12, 13, 17, 18, 19, and 20). Item 1, while it has a loading of 0.42 on the fourth factor, also has a loading of 0.48 on the first factor and was therefore not included in the item analyses. This limitation to those factors with loadings above 0.40 is consistent with

Table 4

Schizophrenia Scales

Test Name (Short Form)	Number of Items for Analysis	Schizophrenia Scale Names
Symptom Rating Scale (SRS)	10	Paranoid Hostility Deteriorated Thinking Unmotivated
Brief Psychiatric Rating Scale (BPRS)	10	Thinking Disturbance Withdrawal - Retardation Paranoid Hostility - Suspiciousness
Symptom-Sign Inventory (SSI)		
- original	10	Paranoid
	10	Nonparanoid
- empirical	2	Paranoid
	7	Nonparanoid
Weighted Symptom-Sign Inventory (WSSI)	1	WSSI
Maine Paranoid Scale	5	Paranoid
	5	Nonparanoid

Table 5

SRS Factor Loadings

Symptom	Uncooperativeness	Depression-Anxiety	Paranoid Hostility	Deteriorated Thinking	Unmotivated
Withdrawn	.48	.26	.07	.42	.32
Evasive-guarded	.79	-.09	.15	.04	.01
Uncooperative	.84	-.09	.20	.18	.10
Lacking rapport	.74	-.06	.14	.21	.22
Disoriented	.11	-.05	.08	.75	.05
Disorganized thinking	.24	-.11	.49	.53	.28
Bizarre postures	.15	-.15	.20	.51	.06
Hallucinatory voices	-.32	.21	.30	.29	.16
Suspicious-paranoid	.12	.01	.68	.19	.18
Manifests depression	.21	.74	-.20	.02	.07
Reports depression	-.08	.82	-.09	-.08	.05
Apathetic	.20	-.07	-.39	.32	.56
Memory deficit	.02	.07	-.21	.70	.03
Manifests anxiety	.11	.42	.38	.08	-.29
Reports anxiety	-.27	.72	.13	-.03	-.13
Physical complaints	-.27	.37	.08	-.03	.11
Lacks motivation	.26	.01	.09	.24	.71
Posthospital goals	-.12	-.18	-.14	.03	-.73
Hospital goals	.09	.09	-.03	-.01	-.64
Excessively hostile	.30	-.09	.65	-.14	-.06

that restriction adopted by Cohen et al. in the interpretation of these five factors.

Brief Psychiatric Rating Scales. John Overall produced the Brief Psychiatric Rating Scales (BPRS) over a number of years and in collaboration with a number of different researchers (e.g., Overall & Gorham, 1962; see Appendix I). The BPRS is composed of 18 seven-point rating scales which refer to terms which were used routinely in the description of psychopathology (e.g., "Conceptual Disorganization"). These ratings could be completed after a brief 15- to 20-minute interview with the patient. Thus, these scales were seen as both clinically meaningful and easy to use.

In an attempt to determine its psychometric properties, Hedlund and Vieweg (1980) reviewed the research employing the BPRS since its inception. They found a median scale interrater reliability of 0.75 which ranged from 0.63 for "Emotional Withdrawal" to 0.88 for "Hallucinatory Behavior". It was also shown to have a certain degree of validity in that changes in scale scores consistently reflected changes in other clinical ratings during treatment.

The endurance of the BPRS is further evidence of its continued prominence. Blashfield (1984) notes that while it only had 23 citations in 1967, its citation has increased steadily with 72 citations in 1979. A recent review of citations indicates that the BPRS continues to be employed frequently for the measurement of schizophrenic symptomatology (e.g., Goff, Henderson, & Amico, 1992; Kuck, Zisook, Moranville, Heaton, & Braff, 1992). Thus, it can be viewed at least as a popular current method of symptom analysis.

This positive support should not indicate, however, that the BPRS is not without criticism. Manchanda, Hirsch, and Barnes (1989) reviewed data on the BPRS and found it wanting in some respects. For example, the items are viewed as broad in what they want to cover and this breadth may lower their usefulness. Also, although interrater reliability may be quite high within a centre, the lack of firm definitions for the seven-point rating scale may result in an idiosyncratic rating system in any particular centre. To guard against this possibility, the results from the present study were compared to previously reported results involving patients diagnosed with schizophrenia.

Several factor analyses have been performed on the BPRS symptom scales and all tend to indicate similar results (Hedlund & Vieweg, 1980). For example, Overall, Hollister, and Pichot (1967) reported the results of several factor analyses of the first 16 BPRS items. Their results indicated four factors which consistently emerged in different samples with different raters in different countries. These four orthogonal factors (and the symptoms with the heaviest factor loadings) were (1) "Thinking Disturbance" (conceptual disorganization, hallucinatory behavior, unusual thought content), (2) "Withdrawal-Retardation" (emotional withdrawal, motor retardation, uncooperativeness, blunted affect), (3) "Paranoid Hostile-Suspiciousness" (hostility, suspiciousness, uncooperativeness), and (4) "Anxious Depression" (somatic concern, anxiety, guilt feelings, tension, depressive mood). The first two of these four factors can be viewed as measures of nonparanoid schizophrenic symptomatology (see Table 4). The third factor can be viewed as a measure of the symptoms

of paranoid schizophrenia. Therefore, these three factors were employed as measures of schizophrenic symptomatology in the present investigation.

Overall et al.'s factor loadings derived from the ratings by psychiatrists of schizophrenics were employed to develop factors of schizophrenic symptomatology (see Table 6). While other sets of factor loadings are available, this particular set best paralleled the underlying population and raters in the present investigation. In a manner paralleling the use of factor loadings in the SRS, the loadings were used in two fashions. To develop the three factor scores, the subject scores on each of the first 16 items of the BPRS were multiplied by the item's factor loading. The 16 resultant scores were totalled separately for each of the three scales. In the analyses that employ only single items, the ten items which have factor loadings of 0.40 or greater were adopted (items 3, 4, 7, 10, 11, 12, 13, 14, 15, and 16).

Other BPRS scales have been devised recently which seek to measure different aspects of schizophrenia. Examples of these scales include: (1) the Paranoid Quotient (Karson & Bigelow, 1986), (2) the Dimension of Schizophrenia (Andersen et al., 1989), and (3) BPRS:NEG (Brier et al., 1987; Thiemann, Csernansky, & Berger, 1987). Nonetheless, scales based on the Overall et al.'s factors continue to be the most widely employed.

Symptom-Sign Inventory. The Symptom-Sign Inventory (SSI) was developed by Foulds (1962, 1965) so that there would be a reliable, quantitative scale available which would be compatable with the psychiatric categories at the time. Foulds wrote ten items, based on the MMPI, research, and clinical experience, for each of the inventory's

Table 6

BPRS Factor Loadings

Symptom	Thinking Disturbance	Withdrawal Retardation	Paranoid Hostility	Anxious Depression
Somatic Concern	.10	-.05	.04	.25
Anxiety	.24	-.11	.22	.71
Emotional Withdrawal	.02	.90	.00	.02
Conceptual Disorganization	.46	.21	.22	-.06
Guilt Feelings	.09	-.09	.04	.73
Tension	.12	.03	.33	.46
Mannerisms-Posturing	.02	.53	.01	-.01
Grandiosity	.27	-.11	.32	-.29
Depressive Mood	.06	.05	-.02	.80
Hostility	-.03	-.06	.88	-.02
Suspiciousness	.30	-.13	.79	.04
Hallucinatory Behavior	.82	.01	-.16	-.02
Motor Retardation	-.07	.65	-.29	.05
Uncooperativeness	-.13	.57	.45	.09
Unusual Thought Content	.82	-.08	.18	-.04
Blunted Affect	.05	.82	-.05	.00

eight scales. The items were written as questions which an experimenter could ask a patient during an interview and were to be answered either "Yes" or "No". The original eight scales were hysteria, anxiety, neurotic depression, obsessionality, mania, psychotic depression, paranoid schizophrenia (and other paranoid states), and nonparanoid schizophrenia. Patients from each of a variety of patient groups were then compared to one another as to their responses on the SSI. To compare nonparanoid and paranoid schizophrenic subjects, a score was derived by subtracting the nonparanoid scale score (see Appendix J) from the paranoid schizophrenia scale score (see Appendix K). On this difference score, the patients with paranoid schizophrenia had an average of 2.87 with a range of 0 to 5. The patients with nonparanoid schizophrenia scored an average of -0.65 and with a range of 1 to -2. According to Foulds, these results indicated that this original paranoid-nonparanoid difference score allowed for adequate subtype separation.

Another possible method of achieving maximum subtype separation is comparing the percentages of the original study's patients from two patient subtypes who responded to an item and then choose the items which originally achieved the best separation (Foulds, 1965). Two lists of 20 such empirically-derived SSI items are included in Appendices L and M. The first list consists of ten questions in which the nonparanoid schizophrenics scored the highest over the paranoid schizophrenics, based on percentages reported by Foulds (1965). The second set contains ten questions in which the paranoid schizophrenics scored higher than the nonparanoids. These two lists are expansions of

previous lists reported by Foulds (1965) and both sets were based on results from his initial research.

In the present investigation, both the original and empirically-derived paranoid and nonparanoid scales were employed as scale measures of paranoid and nonparanoid symptomatology (see Table 4). Also employed was the paranoid-nonparanoid difference score originally envisioned by Foulds as well as a similar score obtained by subtracting the corresponding empirically-derived scales. Those analyses which require only single items (below) employed the 37 items from the two sets of SSI scales.

Weighted Symptom Sign Inventory. Gordon and Gregson (1970) had difficulty with some of the basic theoretical statements made by Foulds. In particular, Foulds (1965) stated that nonintegrated psychotics would perform worse on the SSI than would integrated psychotics. Gordon and Gregson argued that if this was the case, paranoid schizophrenics should be more integrated than nonparanoid schizophrenics. Thus, paranoid schizophrenics should be more symptom-free and have lower scores on the SSI. They compared patients from both subtypes and failed to have such a difference appear.

Gordon and Gregson then put their results through a discriminant analysis to determine which items best separated the two patient subtypes. The function was formed by separately weighting each of the SSI items according to its ability to correctly classify patients as either paranoid or nonparanoid. By multiplying the eleven most discriminating items by their discriminant function weights (see Appendix N), a weighted SSI (WSSI) dimension was formed. This resultant dimension classifies patients as paranoid schizophrenics if they score 5

or more, and as nonparanoid schizophrenics if they score less than 5. This cut-off results in both false-positive and false-negative rates of 17%. Scores from the WSSI have since been employed successfully in investigations for differences between paranoid and nonparanoid schizophrenia (e.g., Neufeld, 1977). Therefore, the WSSI was adopted as an additional schizophrenia scale in the present investigation (see Table 4). Six items in the WSSI were not included in either the original or the empirically-derived scales (F7, C4, F5, H6, B8, and E10) and were, thus, included in the analyses requiring single items.

Maine Paranoid Scale. Written originally by Vojtisek (1976; cited by Magaro, Abrams, & Cantrell, 1981), the Maine Paranoid Scale was developed to separate paranoid and nonparanoid schizophrenics (see Appendices O and P). It includes five items for the measurement of paranoid symptoms and five items for nonparanoid symptoms, all rated on five-point scales. All of these scales are rated by the researcher after a psychiatric interview. Cut-off scores of 12 for the paranoid and 10 for the nonparanoid subscales are suggested for dividing schizophrenics, as long as there was at least a three-point difference between the two scores. For nonschizophrenics, scores below 7 for the paranoid and below 6 for the nonparanoid subscales were considered the most appropriate. While the scores in the middle ranges should be considered schizophrenic, they do not allow for the greatest possible separation between the two subtypes. Thus, individuals with such scores were considered unclassifiable.

Some researchers have reported that this Maine Paranoid Scale classification scheme results in patient classifications which are not consistent with hospital diagnoses. Brennan and Helmsley (1984)

proposed that patients with higher scores on the paranoid scale then the nonparanoid scale should be classified as paranoid. Conversely, if the score is higher on the nonparanoid scale, than the patient would be classified as nonparanoid. Only if the scores on the two scales were equal was the patient unclassifiable. This technique has been employed successfully since it was first proposed (e.g., Lubow, Weiner, Schlossberg, & Baruch, 1987) and was employed in the present study.

Magaro et al. investigated the reliability and validity of the Maine Paranoid Scale through a series of investigations. The four-day test-retest reliability of the nonparanoid and paranoid subscales were 0.73 and 0.89 respectively. The interrater reliability ranged from 0.61 to 0.88. A variety of forms of validity were also assessed. No evidence was found for the two subscales to be correlated. The nonparanoid subscale correlated with measurements reflecting intellectual deterioration (e.g., Embedded Figures Test). The paranoid scale did not correlate with any of these deterioration measures. The subscales did correlate appropriately with other tests for paranoid and nonparanoid schizophrenia (e.g., SSI). Furthermore, neither of the subscales correlated with demographic variables. Factor analysis revealed paranoid and nonparanoid factors (although it should be noted that not all of the scale items loaded on the two factors). Magaro et. al. concluded that, while further work is necessary on the diagnostic capabilities of the Maine Paranoid Scale, it could be used in research as long as it is combined with information from both an interview and from the case records. Researchers have continued to employ the Maine Paranoid Scale and have found that the two scales have

both appropriately correlated with other measures of schizophrenic symptomatology (e.g., Candido & Romney, 1990; O'Reilly et al., 1991).

The present study includes both the paranoid and nonparanoid scales for such scale level analyses (see Table 3). It also included the paranoid-nonparanoid difference as a scale measure. For the item level analysis, all ten items were included.

Scale for the Assessment of Negative Symptoms and

Scale for the Assessment of Positive Symptoms. There were three primary aims in the original development of these scales. First, Andreasen (1982) realized the great utility in the use of negative symptoms in the assessment of the severity of schizophrenia. She noted, however, that no such measure existed at that time. Therefore, Andreasen (1984a) developed the Scale for the Assessment of Negative Symptoms (SANS) based on previous research, including the earlier development of the Scale for the Assessment of Thought, Langauge, and Communication (TLC; Andreasen, 1979a, 1979b).

The present version of the SANS has 25 symptoms or signs (e.g., lack of vocal inflections, poverty of speech, physical anergia) which are rated on six point scales ranging from "none" to "severe" (see Appendix Q). These symptoms and signs are then grouped under five "symptom complexes" which are Affective Flattening or Blunting, Alogia, Avolition-Apathy, Anhedonia-Asociality, and Attention. Each symptom complex is given a brief description in a scoring manual. The description of Alogia, for example, is:

> Alogia is a general term coined to refer to the impoverished thinking and cognition that often occur in patients with schizophrenia (Greek a = no, none; logos = mind, thought).

> Patients with alogia have thinking processes that seem empty, turgid, or slow. Since thinking cannot be observed directly, it is inferred from the patient's speech. The two major manifestations of alogia are nonfluent empty speech (poverty of speech) and fluent empty speech (poverty of content of speech). Blocking and increased latency of response may also reflect alogia. (p. 4)

Each symptom complex contained individual symptom items as well as a global symptom complex rating. Each symptom item included a short definition as well as a more detailed description on how it should be used. For item 1, "Unchanging Facial Expression", the definition and description were:

> The patient's face appears wooden, mechanical, frozen. It does not change expression, or changes less than normally expected, as the emotional content of discourse changes. Since phenothiazines may partially mimic this effect, the interviewer should be careful to note whether or not the patient is on medication, but should not try to "correct" the rating accordingly.
>
> 0 Not at all: Patient is normal or labile
>
> 1 Questionable decrease
>
> 2 Mild: Some decrease in facial responsiveness
>
> 3 Moderate: Facial expressiveness is significantly decreased
>
> 4 Marked: Facial expressiveness markedly decreased
>
> 5 Severe: Facial expression is essentially unchanging (p. 2)

A subscale score was calculated for each complex by adding together its individual symptom items (but not the global rating). A negative

symptom summary score was calculated by adding together the five global ratings. A negative symptom composite score was derived by totalling the 20 individual symptom items. By covering all of these complexes and the many possible symptoms under each, Andreasen (1982) concluded that the SANS filled an important gap in the assessment of schizophrenia.

The second aim in the development of these scales was that there be a complementary scale for assessement of the positive symptoms which occur in schizophrenia. By having a comparison positive scale, research on the range of symptoms would be easier and more meaningful. To this end, Andreasen (1984b) developed the Scale for the Assessment of Positive Symptoms (SAPS). This scale was a modification of the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978). This modification was made so that the SAPS would more closely resemble the SANS.

The SAPS includes ratings of 34 symptoms and signs (e.g., delusions of guilt or sin, repetitive or stereotyped behavior, tangentiality) which are also rated on six point scales ranging from "none" to "severe" (see Appendix R). These symptoms are then grouped into four symptom complexes: Hallucinations, Delusions, Bizarre Behavior, and Positive Formal Thought Disorder. In a manner similar to that described for the SANS, the SAPS scoring manual includes definitions for the symptom complexes, symptoms, and ratings. In addition, several questions are included which the interviewer may wish to use to elicit information on the SAPS symptoms. For example, for "Grandiose Delusions" the interviewer may ask "Are you an unusual person?", "Do you have any special powers or abilities?", and/or "Do you feel you are going to achieve great things?". SAPS subscale scores, a positive symptom

summary score, and a positive symptom composite score are calculated in the same manner as were the SANS scores.

Subsequent research generally has supported the SANS and the SAPS. In reviewing research on the SANS, Andreasen (1989b) listed the reported reliability for the five symptom complexes and concludes that they all display high interrater reliability and internal consistency. Support could also be found for the SANS in its lack of correlation with measures of depression (Morrison et al., 1990; Norman & Malla, 1991) and by its positive correlation with other measures of negative functioning (Jackson et al., 1989). Similar support had been reported for the SAPS (Moscarelli et al., 1987). A recent study of these two scales with 399 patients was conducted by Schuldberg, Quinlan, Morgenstern, and Glazer (1990). They concluded that the interrater reliabilities for the Summary and Composite scales were excellent. The subscale reliabilities ranged from fair to excellent with the lowest reliabilities associated with the more rarely occuring symptoms. Therefore, both the SANS and the SAPS were employed for three purposes in the present investigation. For scale score measurements, the five SANS subscale scores (e.g., alogia) and the four SAPS subscale scores (e.g., hallucinations) were employed. Second, each of the 59 single items from the two scales were included in the item level analysis.

A third aim for the scales was the division of patients into subtypes. Andreasen and Olsen (1982) divided patients into those with Positive Schizophrenia, Negative Schizophrenia, and Mixed Schizophrenia. Positive Schizophrenia was diagnosed in those patients with a score of at least 4 on at least one of the four SAPS global ratings of symptoms complexes and no score of 4 or more on any of the five SANS global

ratings. To be included in the Negative Schizophrenia group, patients needed to have a score of 4 or more on at least two of the SANS global ratings but no score of 4 or more on the SAPS global ratings. The Mixed Schizophrenia category included those patients who did not meet the criteria for either Positive Schizophrenia or Negative Schizophrenia as well as those patients who met the criteria for both. Andreasen and Olsen found that the three groups differed in several ways that were consistent with their proposed nature. For example, the patients with Negative Schizophrenia had lower levels of education than those patients with Positive Schizophrenia. As a result, Andreasen and Olsen concluded that their approach was supported as a useful method of identifying diagnostic subtypes in schizophrenia. The utility of the Positive, Negative, and Mixed Schizophrenia categorization has also been shown to be useful in published research (e.g., Kulhara, Kata, & Joseph, 1986). Nonetheless, while the SANS and the SAPS items and scales have not been changed since they were first introduced, one major change in subtyping criteria has been proposed.

In a recent validation study of these subtypes, Andreasen, Flaum, Swayze, Tyrell, and Arndt (1990) reported that the criteria may have been too restrictive for Negative Schizophrenia. They were changed to a set of criteria which gave greater emphasis to the negative symptoms.

> That is, those patients in the mixed group who had at least two prominent (score of 4 or more) negative symptoms were classified as having negative rather than mixed schizophrenia, based on the assumption that negative symptoms might have greater clinical meaning or predictive validity (p. 616).

This method has since been employed in reseach (Perlata, de Leon, & Cuesta, 1992).

The present investigation employed the revised method for patient categorization. The division of patients into positive and negative subgroups is recognized as secondary in importance in this study compared to their division into paranoid and nonparanoid subgroups. Nonetheless, such divisions would allow for comparing this study's results with those from previous research to assist in the determination of the adequacy of this research.

All of the measurements were taken according to the methods advised by Andreasen (1984b). The subjects were interviewed with respect to symptoms which have been present only in the past month. Andreasen (1984b) emphasized that information should also be derived from other sources such as direct patient observation and the hospital chart. It must be remembered that the emphasis on information gathering was on observations of behavior and not on subjective impressions.

Sternberg Choice Reaction Time Task. While this measure of short-term memory processing (Sternberg, 1966, 1969) is not a symptom scale per se, previous research has indicated that different scores from this task differentiate paranoid schizophrenics, nonparanoid schizophrenics, and normal controls (Broga & Neufeld, 1981). Unfortunately, 39 subjects in this study could not complete the task for several reasons. For many of these subjects, the equipment was not functioning when interview data was being collected. For some others, they did not have their glasses and reported that they were unable to read the digits presented on the screen. For several other subjects, however, the task became too onerous for them to comply with the

instructions for any or all of the length of time necessary to complete it. Therefore, the data from this task were not included in the primary analyses reported in Chapter 3. A description of the task and results of the preliminary analyses are in Appendix V.

Control Measures

Symptom Rating Scale. As mentioned above, five factors have been reported for this scale, three of which can be related to schizophrenic symptomatology (Cohen, Gurel, & Stumpf, 1966). The remaining two scales are not viewed as being related to either paranoid or nonparanoid schizophrenia. These scales, (1) "Uncooperativeness" and (2) "Depression-Anxiety", were adopted as control measures (see Table 7). Consistent with the use of the SRS as a source of paranoid/nonparanoid scales, a patient's scores on each of the twenty items was multiplied by the item's factor loading (see Table 5) and the resultant scores were then totalled to derive the factor score. Also paralleling the above use of the SRS, the control item analyses included those seven items which have loadings of 0.40 on these two factors but not on the other three factors (items 2, 3, 4, 10, 11, 14, and 15).

Brief Psychiatric Rating Scales. One of the four factors which have been found consistently for the BPRS symptom scales (Overall, Hollister, & Pichot, 19967) is not viewed as specifically related to either paranoid schizophrenia or to nonparanoid schizophrenia. This factor, "Anxious Depression", will be employed as a control scale in the present investigation (see Table 7). As with use of the SRS as a source of a control scale, this fourth scale of the BPRS had its score calculated in the same manner as in the development of paranoid and

Table 7

Control Scales

Test Name (Short Form)	Number of Items for Analysis	Control Scale Names
Symptom Rating Scale (SRS)	7	Uncooperativeness Depression Anxiety
Brief Psychiatric Rating Scale (BPRS)	4	Anxious Depression
Symptom-Sign Inventory (SSI) empirical	10	Control

nonparanoid scales. In other words, the subject's score on each of the 16 items was multiplied by the item's factor loadings and then totalled (see Table 5). The control item analysis contained those four items which have factor loadings of 0.40 or greater on this BPRS scale (items 2, 5, 6, and 9).

Symptom-Sign Inventory. Just as paranoid schizophrenia and nonparanoid schizophrenia scales could be empirically-derived by comparing the item response rates of patients from the two subtypes (Foulds, 1965), a control scale was derived by examining the items showing the least difference between groups in response rates. A list of ten such items are included in Appendix S. It should be noted that none of these items were on the original lists of items for either schizophrenic group (see Appendices J and K). Thus, these ten items were also included in the control item analyses.

Procedure

The inpatient procedure for this investigation is outlined briefly in Table 8. Before describing this procedure in detail, two brief comments should be made. First, there were small differences between the inpatient and outpatient procedures for the study: (1) while the inpatients were assessed over two sessions of 30 minutes to 1 hour each, the outpatients were each assessed in only one session in which they completed the WAIS-Clarke IQ Test and the Sternberg Choice Reaction Time Task immediately following the interview, (2) the ratings for the outpatient subjects were made after the completion of the Sternberg Task (not before), and (3) the outpatient chart that was reviewed was not on an inpatient ward but was located in the ATC. Apart from these

Table 8

Outline of Experimental Procedure
(For Inpatients)

PRIOR TO SESSION 1

1) Screen subject a) age between 18 and 60
b) minimum Grade 8 education
c) no evidence of epilepsy
d) no evidence of organicity
e) no electroconvulsive therapy in the last 6 months

SESSION 1

2) Obtain written informed consent

3) Interview subject a) TLC - unstructured speaking
b) TLC - questions
c) SAPS - questions
d) SSI - questions

BETWEEN SESSION 1 AND SESSION 2

4) Score a) SRS
b) BPRS

5) Review chart for previous month a) symptomatology
b) CPZ levels

6) Score a) Maine Paranoid Scale
b) SAPS
c) SANS

SESSION 2

7) Administer WAIS-Clarke IQ Test

8) Administer Sternberg Choice Reaction Time Task

9) Debrief subject

---

differences, the inpatient procedure described below parallels the outpatient procedure.

Second, as noted earlier, three inpatient subjects were not available for the second session. One subject surreptitiously left hospital without signing himself out (i.e., absent without leave). A second subject failed to return from her weekend pass. The third of these subjects signed himself out against physician's advice. All ratings of symptoms were completed after the first session. It was therefore the WAIS-Clarke IQ test and Sternberg Choice Reaction Time Task data that were missing. The majority of analyses could be completed without these data and, as a result, the data from these subjects was retained in the current investigation.

At the beginning of the first session, a signed consent form was obtained which outlined the project's nature and the patient's rights as a potential subject. After signing this form, a semi-structured interview was administered by one of the two interviewers (see below). This interview followed, approximately, the same design as the interview for Thought, Language, and Communication (TLC; Andreasen, 1986). The interview continues to be employed to elicit schizophrenic thought disorder in clinical research with schizophrenics (e.g., Perlata et al., 1992) as well as with other populations (e.g., high functioing autistic adults; Dykens, Volkmar, & Glick, 1991). As can be seen in Appendix T, the TLC interview began with the subject talking about him- or herself for approximately five minutes. The interviewer then asked questions to elict more information about the subject (e.g., "What are the people like whom you work with?"). Because of the dated and foreign political references made in the original Question 4 of the TLC (e.g., President

Reagan's farm policy), this question was changed to the following after four patients were interviewed:

> Tell me about what you think about current political issues like the free trade deal. How do you think Prime Minister Mulroney is doing? Do you think there is a lot of waste in government? What do you think of Mulroney's Goods and Services Tax and the Meech Lake Accord?

In a personal communication, Andreasen (June 14, 1989) concurred with the proposal that such changes were necessary and allowable as long as the types of questions (open versus closed) and the type of content (neutral versus affect-laden) were left the same. Following these TLC questions, the 42 questions from the SSI were asked (see Appendices K, L, M, N, O, and S).

These questions had been placed in a random order and presented to all patients in that same order. Finally, specific questions concerning various positive symptoms were asked. These questions were taken from the SAPS manual (e.g, "Have you heard two or more voices talking with each other?"; Andreasen, 1984b).

The TLC and the SAPS interviews have been completed in the past by individuals such as psychiatrists, fourth year medical students with psychiatric experience (Moscarelli et al., 1987), research assistants with a bachelor's degree in psychology (Andreasen & Grove, 1986), and by master's level research assistants with experience with psychiatric patients (Andreasen, 1982). Because of contextual cues, a live interview is often biased in favour of the subject appearing more understandable than he or she would appear on an audiotape of the same interview. Nonetheless, the live interview is viewed as being the best

in terms of being able to allow raters to most accurately judge negative symptoms such as thought disorder (Andreasen & Grove, 1986). Because interrater reliability is viewed as very important (Andreasen, 1986), the TLC manual stresses that two interviewers should be present at the time of the interview.

With the present interview, the same two raters attended all of its administrations. The first rater was the author who is a master's level student with experience with psychiatric patients. The second interviewer was a research assistant with a bachelor's degree in psychology and with experience in research with psychiatric patients. The two interviewers alternated between patients in the administration of the interview. The other interviewer, at that time, observed the subject and based his or her ratings on the live observation of the interview.

This method, termed the Joint Assessment Method (Spitzer & Williams, 1985), is the most widely used method for ensuring and assessing diagnostic reliability in psychiatry by reducing information variance. It has the added advantage of only interviewing the subject once and, thus, avoiding the need for a second assessment which the patient may not welcome. The Joint Assessment Method has been employed in other research on schizophrenic patients which used the SANS, the SAPS, and the TLC (Andreasen, 1979a; Moscarelli et al., 1987).

Following the interview, the patient returned to the ward. The interviewers then independently completed the SRS and the BPRS. These scales were to be completed on the basis of interview data only. The patient's charts were then independently reviewed by the two raters in order to determine what symptoms were reported by hospital staff during

the past month. Following this review, the Maine Paranoid Scale, the SAPS, and the SANS were independently completed by the interviewers. Data from the charts and the interview were sufficient to answer the questions for all of the scales.

In the second session, the patient first completed the WAIS-Clarke IQ Test. Following this test, the Sternberg Choice Reaction Time Task was administered. After this procedure, each subject was paid for participating ($10.00), signed a receipt book, and was thanked for his or her participation. Care was also taken to answer any questions patients might have had regarding the investigation.

CHAPTER 3 - RESULTS

Adequacy of Present Data

The adequacy of the data from the present study was studied in several ways. The results were investigated as to the level of concordance of the data means and ranges with previous research, each scale's internal consistency and interrater reliability, the correlational analyses among scales, "natural" group differences (male/female, inpatient/outpatient, present/absent history of drug use, acute/chronic, paranoid/nonparanoid hospital diagnosis) on the scales, and scale differences between empirically-derived patient groups. These analyses are reported in detail in Appendix V. It should be noted, at this point, that there was strong support from these various analyses for the adequacy of the present data set by its consistency with previous research in schizophrenia.

Item Reliabilities

Prior to the latent taxonomy analyses of item-level data, an examination of the item reliability was undertaken. Interrater reliabilities were calculable for the items from the SRS, BPRS, Maine Paranoid Scale, SAPS, and SANS (see Table 9). For the 107 items in this listing, 3% were equal to or greater than 0.90, 24% were equal to or greater than 0.80, 50% were equal to or greater than 0.70, 73% were equal to or greater than 0.60, and 90% were equal to or greater than 0.50. Thus, the majority of these items exhibit moderate to good interrater reliability.

Another method of examining these item reliabilities would be to compare them to previously published reliabilities. In their review of the BPRS literature, Hedlund and Verlag (1980) listed the ranges of

Table 9

Item Reliabilities

Item Number	SRS	BPRS	Maine	SAPS	SANS
1	73	62	68	87	67
2	66	46	80	90	63
3	72	74	76	89	62
4	72	80	58	71	73
5	68	59	77	88	59
6	77	56	89	78	71
7	57	71	81	88	69
8	90	84	48	66	74
9	54	59	65	60	80
10	65	52	69	62	59
11	55	55		86	85
12	70	87		73	75
13	35	68		64	67
14	26	80		77	67
15	24	86		85	62
16	65	73		82	58
17	73	71		75	67
18	40	70		84	54
19	53			81	44
20	59			81	58
21				45	59
22				47	74
23				79	50
24				02	90
25				70	69
26				84	
27				77	
28				66	
29				71	
30				61	
31				78	
32				63	
33				85	
34				84	

Note: Decimal points removed

previously reported interrater reliabilities for the first 16 of the 18 BPRS items. The present item reliabilities were within these ranges for 15 of the 16 items. The exception was "Suspiciousness" in which Hedlund and Verlag noted a range of 0.74 to 0.88 and the present study had an interrater reliability of 0.55.

When the SANS item interrater reliabilities were compared to those originally reported by Andreasen (1982), 22 reliabilities were lower than Andreasen's and three were higher. Cortese, Norman, Malla, and Diaz (1992) reported item interrater reliabilities for SANS items 14 to 18. The present SANS interrater reliabilites on these items consistently were higher than those reliabilities reported by Cortese et al. These researchers also reported the interrater reliabilities on the first seven SAPS items. Comparisons with these reliabilities indicated that three were lower than those of Cortese et al. and four were higher. Taken together, these comparisons suggested that the present results for the item interrater reliabilites were sufficiently consistent with previous research to support their continued use in item-level analysis.

Formation of Variable Clusters

Because of the large number of schizophrenia variables (122) in relation to the number of subjects (100), attempts were made to make it more manageable for future analyses. Such "judicious pruning" of large data sets was felt to be advisable in such circumstances to improve the quality of information and clarify the outcome (Kraemer et al., 1987). The method followed closely paralleled that outlined by Kraemer (1984) in her reanalysis of the schizophrenic symptomatology profiles from the subjects in the International Pilot Study of Schizophrenia. There were

three separate steps in Kraemer's reanalysis: (1) Directionality of items (Rescoring items), (2) Deletion of items, and (3) Removal of redundancies. These steps were undertaken separately for the schizophrenia items and for the control items.

The first step, directionality of items, was undertaken to ensure that all items were scored in a uniform direction. Such uniformity would ensure that all items were measuring the same underlying construct in a similar fashion. Thus, the directionality of any future covariances between any two pairs of items would be consistent in what they indicated.

To accomplish this end, item-total correlations were calculated. All of the items which had negative correlations with the total were rescored in the opposite direction. For the schizophrenia data, SRS items 18 and 19 were reversed. Since such rescoring affected all of the item-total correlations, these calculations were repeated. On the second trial, the schizophrenia item-total correlations were uniformly positive. For the 21 items of control data, SSI items 10 and 27 had to be reversed on the first set of item-total correlations because of their negative correlations. On the second trial, SSI items 22 and 27 had negative item-total correlations and were reversed. On the third trial of correlations, all of the items were positively correlated with the total score.

The second step, deletion of items, was undertaken to ensure that all of the items were related to the underlying disorder. The last set of schizophrenia item-total correlations was reviewed and all of those correlations which failed to exceed a level expected by chance were not included in any further analyses. For these schizophrenia items,

25 items were removed at this stage (SRS 13, 18, 19, 20; SSI 04, 09, 11, 12, 13, 17, 21, 23, 24, 25, 35, 38; SAPS 09, 10, 24, 30, 31; SANS 02, 09, 19, 24). The item-total correlations were recalculated with the 97 remaining items and two additional items were then removed since they were not significant (SSI 15, 40). On the third trial of these correlations, all 95 of the remaining schizophrenia items were significant. A general listing of the items deleted is in Table 10.

It should be noted that the items for the control profile did not go through this second step. These items were chosen because they were not related to schizophrenia or did not differentiate the paranoid and nonparanoid subtypes. Thus, they would not be related to any specific latent trait. There would then be no use in deleting a control item at this stage of analysis if it failed to be related to the remainder of control items.

Kraemer's third step was to reduce redundancies. Several of the schizophrenia items from different measures were designed to tap into the same construct. For example, BPRS item 8 "Grandiosity" and SAPS item 11 "Grandiose Delusions" would overlap to a significant degree. A negative symptom example would be that of SRS item 12 "Is the patient apathetic?" and SANS item 17 "Global rating of avolition-apathy". Such an overlap would result in a high level of multicollinearity among the variables. Such multicollinearity could have had a severe negative effect on future analyses. Combining items which tapped similar constructs allowed for a smaller number of variables and reduced the possibility of problems resulting from multicollinearity (e.g., Brockington, Kendell, Wainwright, Hillier, & Walker, 1979).

Table 10

Schizophrenia Items Removed Prior to Cluster Analysis
(Page 1 of 2)

SRS 13 - Pathological memory deficit

SRS 18 - Specificity of posthospital goals

SRS 19 - Specificity of hospitalization goals

SRS 20 - Evidence of excessive hostility

SSI 04 - Are you excessively concerned about cleanliness

SSI 09 - Is there something unusual about your body

SSI 11 - Do you ever have very strange and peculiar thoughts at times

SSI 12 - Have you ever attempted to do away with yourself

SSI 13 - See something that you know has a special meaning

SSI 15 - See things around you that other people don't seem to see

SSI 17 - Have you an important mission to carry out

SSI 21 - Do you ever wonder who you really are

SSI 23 - Are you more absent-minded recently than you used to be

SSI 24 - Cannot communicate with others because not on same wavelength

SSI 25 - Have you lost interest in almost everything

SSI 35 - Times when exciting new ideas occur to you one after another

SSI 38 - Puzzled something wrong with you or world but don't know what

SSI 40 - Do you think people regard you as odd

Table 10

Schizophrenia Items Removed Prior to Cluster Analysis
(Page 2 of 2)

SAPS 09 - Delusions of jealousy

SAPS 10 - Delusions of guilt or sin

SAPS 24 - Repetitive or stereotyped behaviour

SAPS 30 - Circumstantiality

SAPS 31 - Pressure of speech

SANS 02 - Decreased spontaneous movements

SANS 09 - Poverty of speech

SANS 19 - Sexual activity

SANS 24 - Inattentiveness during mental status testing

The method employed by Kraemer (1984) for combining items to reduce multicollinearity was cluster analysis. In the present investigation, hierarchical cluster analyses for variable clustering from SPSS-X was undertaken. These analyses were undertaken for the schizophrenia items and for the control items separately. These procedures employed Ward's method (Ward, 1963; Ward & Hook, 1963) as it has been supported as the most consistently valid under a variety of conditions (Meehl, 1992; Milligan & Cooper, 1987; Mojena, 1977).

The variables, however, were on scales of different magnitudes, ranging from binary (Yes/No) in the SSI to a seven-point scale in the BPRS. Such differences in magnitude affect many measures of dissimilarity, including Euclidean distance, and should be standardized (Fleiss & Zubin, 1969). In reviewing the methods for the standardization of variables under a variety of different circumstances, Sneath and Sokal (1973) recommended Gower's (1971) method of ranging when dealing with both two-state and multi-state variables. In this method, the smallest value for the variable ($\underline{x}$) was subtracted from the value and the result was divided by the range:

$$\underline{z} = (\underline{x} - \text{Min}(\underline{x})) \; / \; (\text{Max}(\underline{x}) - \text{Min}(\underline{x})).$$

In a Monte Carlo study of the standardization of variables in cluster analysis, Milligan and Cooper (1988) compared seven methods. They reported that "apparently, standardization by division by the range of the variables consistently aids in cluster recovery and is robust under a variety of conditions" (p. 202). Therefore, this method was employed for the two sets of items in the study.

There was no pre-determined number of clusters that was thought to be appropriate for the present data. The number of clusters finally

accepted depended on the results. Milligan and Cooper (1985) employed a Monte Carlo evaluation to examine 30 procedures for an empirically-based determination of the number of clusters in a given data set. The best of these methods, the Calinski and Harabasz index (1974), was adopted in an attempt to determine what would be the most accurate number of clusters.

Calinski and Harabasz termed this index the Variance Ratio Criterion (VRC). The VRC is analagous to the F-statistic in univariate analysis because it is concerned primarily with the within-group (cluster) sum of squares (WGSS) and the between-group (cluster) sum of squares (BGSS). The total sum of squares (TSS) was defined as:

$$TSS = \sum_{x=1}^{n-1} \sum_{y=x+1}^{n} d2_{(x,y)}$$

where: n = the total number of variables, and

d = the distance between two variables.

Also defined was the Total Sum of Squares for each cluster (TSS_c) in a group of clusters:

$$TSS_c = \sum_{x=1}^{m-1} \sum_{y=x+1}^{m} d2_{(x,y)}$$

where: c = number of that cluster,

m = the number of variables in that cluster, and

d = the distance between two variables.

The WGSS was then defined as:

$$WGSS = \sum_{c=1}^{k} TSS_c,$$

where: k = the total number of clusters.

As with the F-statistic, the TSS was the sum of the BGSS and the WGSS. Thus, the BGSS was calculated by:

$$BGSS = TSS - WGSS.$$

The VRC was then defined, in a manner similar to the F-statistic, for each possible number of clusters:

$$VRC = \frac{BGSS}{k-1} \Big/ \frac{WGSS}{n-k},$$

where: k = total number of cluster,

n = total number of variables.

Calinski and Harabasz suggested choosing that number of k for which the VRC has an "absolute or local maximum". If there were more than one local maximum, they suggest that it may be the most economical to choose the smallest value of k although other values of k may also be valid. If there was a steady lessening of the VRC, they suggested that the maxima from the first increase be employed.

While not a widely employed statistic, use of the VRC has had some important precedent in research. In psychology, recent cluster analysis research has employed the VRC in investigations of children's understanding of emotions (Reissland, 1985), alcohol- and drug-abuse histories in incarcerated offenders (Hodgins & Lightfoot, 1988), alcoholic personalities (Retzlaff & Bromley, 1991), and non-specific low

back pain (Coste, Spira, Ducimetjere, & Paolaggi, 1991). Other disciplines as diverse as botany (Baum, Tulloch, & Bailey, 1989), economics (Downes, 1990), and climatology (Stooksbury & Michaels, 1991) have also employed the VRC in cluster analysis research. Thus, it is supported as a useful measure in cluster analysis research.

The VRCs for the schizophrenia data and the control data are listed in Table 11. The schizophrenia data VRCs steadily declined from 2 clusters to 25 clusters and had a local maxima at cluster 26. It was therefore decided, as per Calinski and Harabasz (1974), that the data be divided into 26 "symptom clusters". (It is recognized that many of the "clusters" only contained one item. They are termed clusters to allow for simplicity of the discussion in the following sections.)

The control data displayed steadily declining VRCs from 2 "clusters" to the maximum 21 "clusters" (which represented each individual item). Thus, these data indicated that the control items were either a uniform cluster or did not cluster at any level. Given the wide disparity of items chosen for analysis and that these items had no common theoretical core, the data were not assumed to form a single cluster. Therefore, further control variable "cluster"-level analysis only employed data at the item level.

Once the nature and number of clusters was identified, the data were aggregated within these clusters to form a symptom subscale (see Appendix W for a listing of the items within the schizophrenia clusters). There is some controversy as to whether such data aggregation should involve standardized data (cf. Gardner & Erdle, 1984, 1986; Stevens & Aleamoni, 1986). While it was recognized that the aggregation of raw scores is sometimes more appropriate (Gardner &

Table 11

Calinkski and Harabasz
Variance Ratio Criteria (VRC)
for Schizophrenia and Control Data

Number of Clusters	Schizophrenia Index	Control Index
2	917490	455321
3	804367	414946
4	659607	371817
5	577732	344748
6	520932	326931
7	520932	304943
8	442654	287156
9	416608	273199
10	395027	262103
11	377321	250893
12	361233	240130
13	346825	231095
14	334249	222549
15	323231	211367
16	312682	201589
17	305181	192031
18	296550	183567
19	288615	175385
20	279380	167609
21	268017	160447
22	262476	
23	256682	
24	251319	
25	241344	
26	246204 *	
27	243311	
28	238549	

* First local maxima following decline from 2 cluster solution

Note: Decimal points removed

Erdle, 1984), standardized data were aggregated in the present study. The reasons for standardization in this case were two-fold. First, the cluster analyses were based on such standardized data. Second, there do not seem to be any a priori reasons to believe that a BPRS item (on a seven-point scale) with greater range and variability should be given more weight in the aggregate than would a SSI item (on a two-point scale).

Two additional points should be made with regard to these analyses. First, the present study employed 95 symptom variables from different measures and the resultant cluster analysis indicated 26 clusters. It is interesting to compare these figures to comparable figures from the International Pilot Study of Schizophrenia (WHO, 1973). In that study, there were 124 units of analysis (general symptoms). These symptoms then were grouped together into major areas of dysfunction based on clinical judgement. This reduction resulted in 27 groups of units of analysis. Some of their groups of units were very similar to those described in this investigation (e.g., Group 7: "Experinces of Control" and Symptom Cluster 4: "Thought Control"). Other groups were condensations or subdivisions of the present clusters (e.g., Group 3: "Quantitative Disorder of Form of Thinking (and Speech)" and Group 4: "Qualitative Disorder of Form of Thinking (and Speech)" compared to Cluster 17: "Bizarre Behaviour/Language" and Cluster 18: "Disorganized Thought"). Thus, while there are many differences in the resultant clusters, it is interesting to note that similarly large variable sets were reduced to a similar number of clusters.

Second, the point should be mentioned that it is recognized that the SSI items tended not to cluster with the items from the other

scales. This tendency may have resulted from the different variances present with the SSI items in comparison to the other items. Thus, because of the more similar pattern in their variances, the other items would tend to cluster more. Nonetheless, the SSI items did cluster with the SAPS on one occasion (Symptom Cluster 4 - "Thought Control"). Thus, while the SSI items may not have correlated strongly with the other items, they were not totally isolated. In addition, there was no evidence that single-item clusters (e.g., Symptom Cluster 2 - "Faultless Criticism") were not valid for the taxometric analyses. Since the symptom clusters were to be put through further consistency tests to determine their adequacy for the taxometric analyses, the decision was made to retain these clusters for fear of prematurely removing items which might assist in the subsequent analysis.

Definitions of Clusters

The next stage of the analysis involved determining which of the clusters were measures of paranoid symptomatology, nonparanoid symptomatology, positive symptomatology, and negative symptomatology. To achieve this determination, ratings as to the nature of the symptoms were solicited from three individuals (two Ph.D. level clinical psychologists and one psychiatrist). These raters were all involved in research in schizophrenic symptomatology, were not working together on any research projects at that time, and were not involved in the analysis of the data in this study.

The researchers were given a listing of the items within each cluster. They were also asked to rate these clusters in two manners. First, they were to rate each of them as Paranoid, Nonparanoid, or Both/Neither (see Table 12). Second, they were to rate each cluster as

Table 12

Cluster Ratings for
Paranoid and Nonparanoid Status

Cluster Number	Rater 1	Rater 2	Rater 3	Decision
1	-	-	-	-
2	P	P	-	P
3	-	-	-	-
4	-	P	P	P
5	-	-	-	-
6	-	P	-	-
7	P	-	-	-
8	-	-	-	-
9	P	P	P	P
10	N	N	-	N
11	N	N	-	N
12	N	-	-	-
13	-	N	-	-
14	N	N	-	N
15	P	P	P	P
16	P	P	-	P
17	N	N	-	N
18	N	N	-	N
19	P	P	-	P
20	N	N	-	N
21	N	N	-	N
22	-	-	-	-
23	-	N	N	N
24	-	-	-	-
25	N	N	-	N
26	N	N	N	N

P = Paranoid (6 clusters)

N = Nonparanoid (10 clusters)

- = Both/Neither (10 clusters)

Positive, Negative, or Both/Neither (see Table 13). One rater only rated five of the 26 clusters as Paranoid or Nonparanoid and rated the remaining 21 clusters as Both/Neither. Therefore, it was decided that if two of three raters agreed on a classification and the third did not rate the cluster in the opposite grouping (e.g., two ratings as Paranoid and the third rating as Nonparanoid), then it would be classified as per these two raters. As Table 12 indicates, six clusters were then rated as Paranoid, 10 as Nonparanoid, and 10 as Both/Neither. Table .3 lists how 12 symptoms were classified as Positive, four as Negative, and 10 as Both/Neither. Overall, six clusters of data were not classified and were not included in any further analysis (Clusters 1, 3, 5, 7, 8, and 12). After being classifed, each of the remaining 20 clusters were labelled to indicate the general types of items included in it (see Table 14).

Consistency Tests

In discussing how data should be prepared for any analysis of latent structure, Meehl (1992) wrote "I cannot emphasize too strongly that, in my view, multiple consistency tests are a desideratum" (pp. 138-139; italics in original). They were not seen as merely useful extras to Meehl. He gave his reasoning for them as follows:

> Since taxometrics (like factor analysis) is a bootstraps procedure, forced to rely on the internal pattern of relations among fallible indicators (having no external defining criterion), any taxometric method that lacks consistency tests is radically defective (p. 139; original italicized).

Meehl's investigations of these consistency tests were a part of his larger 1970's research on detecting latent clinical taxa. This

Table 13

Cluster Ratings for
Positive and Negative Status

Cluster Number	Rater 1	Rater 2	Rater 3	Decision
1	-	-	-	-
2	-	P	-	-
3	-	-	-	-
4	P	P	P	P
5	-	-	-	-
6	P	P	-	P
7	P	-	-	-
8	-	-	-	-
9	-	P	P	P
10	P	P	P	P
11	-	P	-	-
12	-	P	-	-
13	-	P	P	P
14	P	N	-	-
15	P	P	P	P
16	-	P	-	-
17	P	-	P	P
18	P	P	P	P
19	P	P	P	P
20	N	-	N	N
21	N	-	N	N
22	P	P	P	P
23	N	N	N	N
24	P	P	-	P
25	P	P	-	P
26	N	N	N	N

P = Positive (12 clusters)

N = Negative (4 clusters)

- = Both/Neither (10 clusters)

Table 14

Summary of Clusters and Titles

Cluster Number	Paranoid/ Nonparanoid	Positive/ Negative	Title
1			NONE
2	Paranoid		Faultless Criticism
3			NONE
4	Paranoid	Positive	Thought Control
5			NONE
6		Positive	Illness/Madness Fear
7			NONE
8			NONE
9	Paranoid	Positive	Conspiracy Fear
10	Nonparanoid	Positive	Realization of Strange Thoughts
11	Nonparanoid		Sex/Religion Distressing Thought
12			NONE
13		Positive	Strange Voices
14	Nonparanoid		Dream-Like State
15	Paranoid	Positive	Special Powers
16	Paranoid		Delusions of Persecution
17	Nonparanoid	Positive	Bizarre Behaviour/Language
18	Nonparanoid	Positive	Disorganized Thought
19	Paranoid	Positive	Delusions
20	Nonparanoid	Negative	Apathy
21	Nonparanoid	Negative	Inappropriate Affect/Inattention
22		Positive	General Hallucinations
23	Nonparanoid	Negative	Emotional Expression Blunting
24		Positive	Aggressive Behaviour
25	Nonparanoid	Positive	Clanging
26	Nonparanoid	Negative	General Negative Symptoms

research was published as a series of internal research reports in the Department of Psychiatry at the University of Minnesota. Two of these reports specifically centred on the consistency tests (Golden & Meehl, 1973; Golden, Tyan, & Meehl, 1974). The consistency tests employed in the present investigation were the three tests suggested by Golden and Meehl (1979).

Two general points should be briefly outlined regarding these analyses. First, MAXCOV and the related consistency tests are most easily employed with dichotomous data. All of the previous research employing these techniques have employed dichotomous data. By employing the statistics on a small group of dichotomous variables, it is possible to employ the statistics without the necessary specialized computer programming that is not yet widely available. Therefore, each of the variables were dichotomized (0, 1) as close as possible to their medians (Grove et al., 1987). Second, this present discussion will focus on the Positive Symptom Clusters but only for illustrative purposes only. All five of the variable groups, or groups of symptom clusters (paranoid, nonparanoid, positive, negative, and control), had the same series of analyses performed.

Consistency test 1. One consistency test suggested by Golden and Meehl (1979) involved initially creating scales within each variable group. At this initial point of the analysis, there were 12 Positive Symptom Clusters. These clusters were used to develop 12 scales. For scale 1, the scores on clusters 2 through 12 were totalled. For scale 2, the scores on clusters 1 and 3 through 12 were totalled. This pattern continued until all 12 possible scales had been developed. Each of these 12 scales had a theoretical range of scores from 0 to 11 (in

this particular instance, no scale had a score of 0 so the actual range was from 1 to 11).

The next stage was to examine each Positive Symptom Cluster using the formula:

$$\underline{d}(\underline{c}) = \underline{a}(\underline{c}) - \underline{b}(\underline{c}),$$

where $\underline{a}$ = mean symptom cluster score for subjects scoring above $\underline{c}$,

$\underline{b}$ = mean symptom cluster score for subjects scoring below $\underline{c}$,

$\underline{c}$ = a score (range 0 to 11) on the cluster's corresponding scale,

$\underline{d}$ = mean difference between subjects scoring above and below $\underline{c}$.

Thus, each cluster theoretically had 12 values each (one for each of the the scores 0 to 11 of the scale) for $\underline{a}$, $\underline{b}$, and $\underline{d}$. The Positive Symptom Cluster 4 scale scores for these values for each level of $\underline{c}$ are presented in Table 15.

To control for any fluctuations due to sampling error in the data, curve smoothing (see below) of the $\underline{d}$ was suggested by Golden and Meehl (1979) before any decisions were made regarding the data. After the curve smoothing, any symptom cluster from a variable group with a maximum $\underline{d}$ of less than 0.10 was eliminated from further analyses and this first consistency test was reattempted. According to Golden and Meehl, this criterion is based on results from the analyses of several artificial data samples. The results of Golden and Meehl's analyses indicated that further taxommomic analyses would not give accurate results if the maximum $\underline{d}$ was not at least at a level of 0.10. Finally, this consistency test and the next two to be described were iterative in that they were reattempted continually until no further items were eliminated.

Table 15

Consistency Test 1 Example:
Positive Scale - Cluster 4 - First Attempt

Scale Score	Above	Below	Difference
1	.53	---	(.53)
2	.64	.10	.54
3	.71	.04	.67
4	.79	.13	.66
5	.83	.17	.66
6	.90	.26	.64
7	.94	.31	.63
8	.92	.40	.62
9	1.00	.43	.57
10	---	.47	(.53)
11	---	.49	(.51)

Curve smoothing. This set of techniques has been developed for uncovering patterns in data with a minimum of assumptions regarding the nature of the underlying pattern. The technique's primary assumption is that the best relations between variables are smooth. Tukey (1977) described the relation:

$$\text{data} = \text{smooth} + \text{rough},$$

as being the same as:

$$\text{data} = \text{fit} + \text{residuals}.$$

Cohen (1990) has endorsed the use of such graphic representations of data for exploratory data analysis.

Tukey describes a number of possible smoothing techniques that could be combined in several manners but Meehl (1973) proposed that the "3RH Twice" method be employed in MAXCOV analyses. It has since been used by a number of researchers employing MAXCOV (e.g., Gangestad & Snyder, 1985). Such techniques, which include "running odd number medians" and "hanning" (see below) were recommended because of their "common sense criteria", their "theoretical justification", "and "their proven effectiveness" by Goodall (1990) in a review of specific curve smoothing techniques.

Table 16, based on the raw difference data from the first consistency test on Positive Symptom Cluster 4 will be used to illustrate the "3RH Twice" technique. First, "raw" refers to the "Difference" scores from Table 15. In the next column, the "3" refers to "running medians of 3". Three successive difference scores were arranged in order of value. Next, the middle (i.e., median) value was written down across from the middle value of the sequential order. For example, with scale scores 2, 3, and 4, the raw difference scores were

Table 16

Consistency Test 1
(Curve Smoothing) Example:
Positive Scale - Cluster 4 - First Attempt

Scale Score	Raw	Once			Twice		
		3R	)	H	3R	)	H
1	53	(53)		(53)	(53)		(53)
2	54	54	59h	57	57	58	58
3	67	66	60	63	63	61h	62
4	66	66	66	66	66	64h	65
5	66	66	65	66	66	65	66
6	64	64	64h	64	64	64h	64
7	63	63	63h	63	63	63h	63
8	62	62	60	61	61	60	61
9	57	57	57h	57	57	57h	57
10	53	53	54	54	54	54	54
11	51	(51)		(51)	(51)		(51)

(number) = "copied on"

h = half

Note: decimal points removed

54, 67, and 66 respectively. These scores were then arranged in order of value (54, 66, 67) and the median value (66) was chosen. It was then written beside the middle value of the sequential order (67). These "running medians of 3" continued for all groups of three sequential items. "3R" represents repeating such "smoothings" until there is no further change. Such a pattern was evident in Table 16 after only one smoothing.

The end values for scale scores 1 and 11 were then "copied on" according to Tukey's extrapolated end value rule. Tukey described this rule as such: "We look at two differences:

end input-value MINUS next-to-end smoothed value

and

next-to-end smoothed value MINUS next-to-end-but-one smoothed value

and if the first is between 0 and +2 times the second, we can copy on" (p. 221). At the top of Table 16, the rule allows us to "copy on" the value of 53 for scale score 1.

If the rule did not allow us to "copy on" a value, then another method is employed. To determine what value could be added, Tukey's extrapolation technique was employed when necessary. With this technique, the end value was calculated by using the following formula:

(end input-value MINUS next-to-end smoothed value) =

2(next-to-end smoothed value MINUS next-to-end-but-one smoothed value)

In his review of these smoothing techniques, Goodall (1990) supported these end-value techniques for use with 3R in exploratory data analysis.

Next, the "H" in "3RH Twice" refers to "hanning". The first step in hanning is to form "skip means" (listed as ")" in Table 16). With

this step, the mean of two values was calculated. These two values were from one line above and from one line below the line on which the skip mean was entered. For example, the first skip mean for c = 3 was 60 which was the mean of the 3Rs for c = 2 (54) and c = 4 (66).

In the next step in hanning, the line mean (H) was calculated as the mean of (i) the 3R and (ii) the skip mean for a given c. For example, when c = 3, the first 3R = 66 and the first) = 60, which resulted in a first line mean (H) of 63. This process was then continued for the whole range of c (with the exception of the end values where the extrapolated end value rule was once again applied).

The "Twice" in "3RH Twice" refers to a repetition of the 3RH. "Compound smoothers", such as "3RH Twice", result in curves which have often been viewed as "needed to properly analyze the data" (p. 143; Goodall, 1990). Table 16 lists both the first smoothing and the compound (i.e., second) smoothing.

Figure 9 presents the curves from the raw data, initial smoothed data, and final smoothed data. This figure illustrates how the curve becomes progressively smoother. In the present investigation, these techniques were employed for each variable (i.e., symptom cluster) on each trial of Consistency Test 1 for all five variable groups.

Consistency test 2. The second consistency test revolves around the "hitmax cut". In this instance, the hitmax cut was the scale score that best maximizes the total number of correct classifications if subjects were to be divided into (in this illustration) positive and non-positive subjects. Golden and Meehl (1979) discuss how the hitmax cut is the cutting score on the scale score variable that would be associated with the intersection of the positive subject frequency

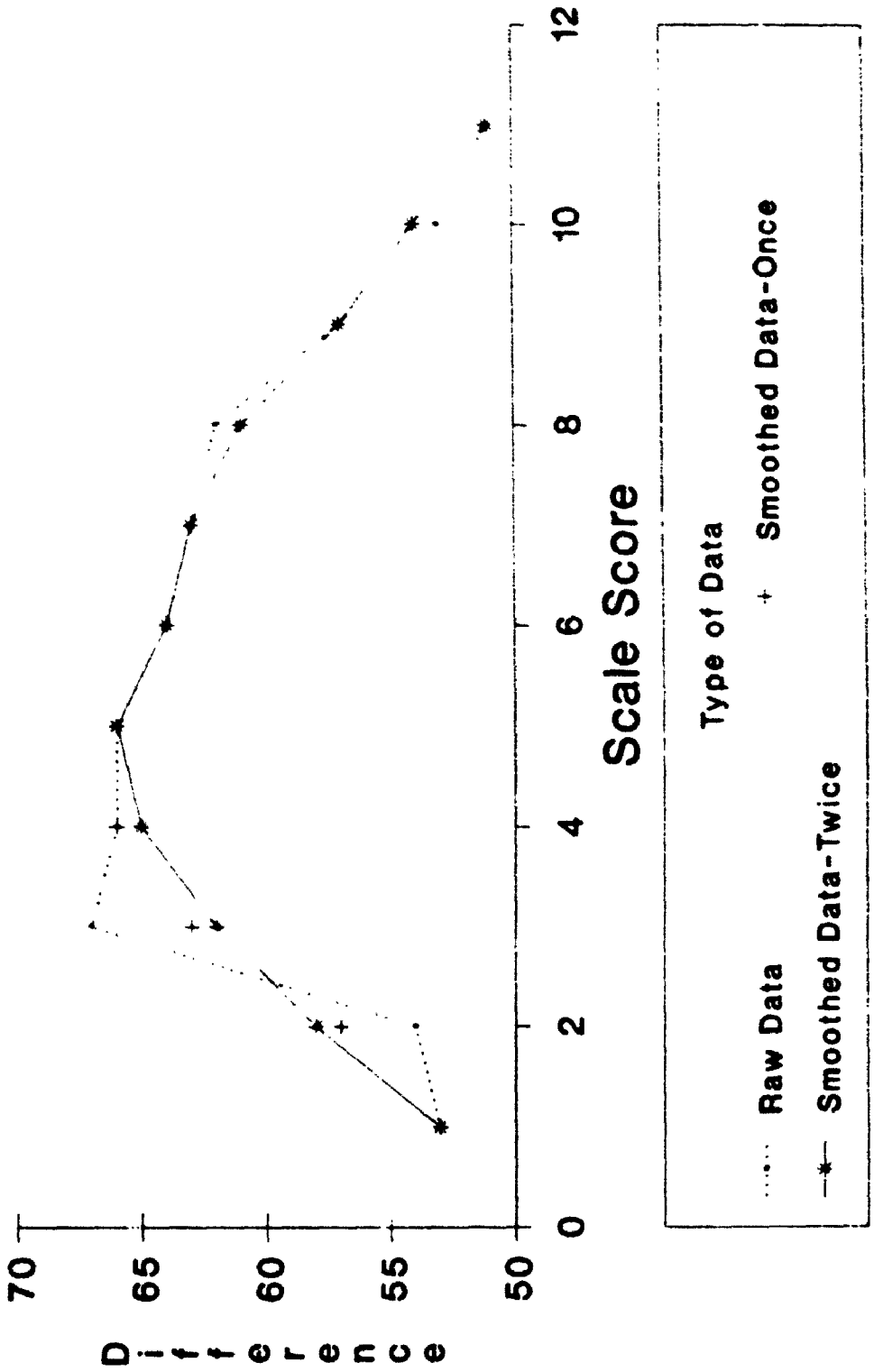
FIGURE 9

Curve smoothing example:

Consistency test 1 for

Positive Scale Cluster 4

- First attempt


Difference
70
65
60
55
50
0
2
4
6
8
10
12
Scale Score
Type of Data
Raw Data
Smoothed Data-Once
Smoothed Data-Twice

distribution and the non-positive subject frequency distribution. Golden and Meehl (1973) reported that the value of c (scale score) corresponding to the maximum of the smoothed d (difference) curve was a very good estimate of h (hitmax cut).

This second consistency test compares the h of each variable (symptom cluster) to the mean h for all of the variables (symptom clusters in a variable group). To pass this test, Golden and Meehl (1979) advised that a variable's h must be within 15% of the total range around the variable group mean h. Golden and Meehl (1973) reported that Monte Carlo studies of this consistency test indicated that subsequent latent taxonomy analyses could tolerate that level of variability in the estimated hitmax cut.

For Positive Symptom Cluster 4 at the third trial of this second consistency test, h = 2. The mean h for the Positive Variable Group was 3.0. Since the range of scores was 8 (scale scores 0 through 7), the acceptable range was 1.8 through 4.2 (3 + or - 1.2). Therefore, this symptom cluster passed this trial of the second consistency test and could continue with the variable group to the next trial or to the third consistency test.

Consistency test 3. For this final consistency test, each subject had a scale score calculated to correspond with each variable (symptom cluster) in a variable group. This score was calculated by totalling the scores on the remaining variables in that variable group. The subjects were then ranked according to this scale score for each variable. According to this ranking, the top quartile of subjects in each variable were viewed as being the most representative of that variable group. Subjects in the bottom quartile were seen as being the

best to represent the opposite of that variable group. According to Golden and Meehl (1979), if the individual variable was related to the variable group, than the difference between the upper and lower quartile subject groups in their individual-variable scores should be at least 0.15 and preferrably at least 0.20. Golden and Meehl (1973) reported that Monte Carlo investigations supported this difference as appropriate as a screen for variables continuing to further analyses.

As an illustration of consistency test 3, the scores on Positive Symptom Clusters 6, 9, 13, 15, 17, 18, and 24 were totalled to develop a scale score for evaluating Positive Symptom Cluster 4. The top quartile of individual subjects on this scale were thought to be Positive Symptom Cluster 4's best representatives of positive subjects (p(Pos)). Conversely, the bottom quartile were thought to best reflect the non-positive subjects (p(Non)). These two groups (i.e., quartiles) were then compared in their average score on Positive Symptom Cluster 4. The mean score for the top quartile was 0.86 while the bottom quartile mean was 0.13. Thus, the difference score for Positive Symptom Cluster 4 was 0.73. Since this difference score exceeded the minimum difference necessary (0.15), Positive Symptom Cluster 4 passed this trial of Consistency Test 3. As Table 17 indicates, each of the eight surviving Positive Symptoms Clusters passed this third consistency test.

Table 18 reviews the results of these three consistency tests. The control items were put through these tests because, if they were to be comparable after the MAXCOV analysis, they must have passed through the same required consistency hurdles as the schizophrenia symptoms. The usual number of items for such analyses in previous research was usually eight although there are no firm rules for the number of items required.

Table 17

Consistency Test 3 Example:
Positive Clusters

Cluster Number	p (Pos)	p (Non)	Difference
4	86	13	73
6	69	4	65
9	79	11	68
13	65	26	39
15	57	0	57
17	80	10	70
18	77	32	45
24	88	22	66

Note: all positive clusters passed consistency test 3 (i.e., Difference >= 15)

Note: decimal points removed

Table 18

Summary of Results of Consistency Tests

Test	Paranoid	Nonparanoid	Positive	Negative	Control
Prior	6	10	12	4	21
Test 1					
- 1st Trial	6	9	11	4	20
- 2nd Trial		8	11		20
- 3rd Trial		8			
Test 2					
- 1st Trial	6	8	9	4	15
- 2nd Trial			8		14
- 3rd Trial			8		14
Test 3					
- 1st Trial	6	8	8	4	11
- 2nd Trial					10
- 3rd Trial					10
Internal Reliability	0.80	0.67	0.79	0.73	0.62

The internal reliabilities (measured at the symptom cluster level) of the resultant groups were generally good with the lowest being with the control group of item-level variables (Cronbach's alpha = 0.62). The final set of schizophrenia symptom clusters are listed in Table 19 and the final set of control items are listed in Table 20.

Consistency test symptom cluster removals. The Paranoid and Negative groups of symptom clusters did not lose any of their member clusters as a result of the consistency tests and will not be discussed further in this section. The Nonparanoid Variable Group lost two symptom clusters as a result of the three consistency tests. One of these two clusters (Symptom Cluster 25 - SAPS item 33 - "Clanging") had a low prevalence in the study's patient sample. The resultant low variability would have resulted in low covariation and would have made group differences more difficult to detect. This lack of significant variability may have been at the root as to why it did not behave similarly to the other Nonparanoid Symptom Clusters and, therefore, did not pass the consistency tests.

The reasons are not as clear as to why the other removed Nonparanoid Variable Group Symptom Cluster (Symptom Cluster 23 - "Emotional Expression Blunting") did not pass the consistency tests. The Nonparanoid Variable Group includes the other three of the four Negative Variable Group Symptom Clusters. That Variable Group also includes the other three symptom clusters from the Negative Variable Group. Thus, the reasons for its exclusion is probably more related to a specific aspect of the symptom cluster itself and not to the general issue of negative symptomatolgy in the Nonparanoid Variable Group.

Table 19

Schizophrenia Scale Clusters

Paranoid

Cluster 2 - Faultless Criticism
Cluster 4 - Thought Control
Cluster 9 - Conspiracy Fear
Cluster 15 - Special Powers
Cluster 16 - Delusions of Persecution
Cluster 19 - Delusions

Nonparanoid

Cluster 10 - Realization of Strange Thoughts
Cluster 11 - Sex/Religion Distressing Thoughts
Cluster 14 - Dream-Like State
Cluster 17 - Bizarre Behaviour/Language
Cluster 18 - Disorganized Thought
Cluster 20 - Apathy
Cluster 21 - Inappropriate Affect/Inattention
Cluster 26 - General Negative Symptoms

Positive

Cluster 4 - Thought Control
Cluster 6 - Illness/Madness Fear
Cluster 9 - Conspiracy Fear
Cluster 13 - Strange Voices
Cluster 15 - Special Powers
Cluster 17 - Bizarre Behaviour/Language
Cluster 18 - Disorganized Thought
Cluster 24 - Aggressive Behaviour

Negative

Cluster 20 - Apathy
Cluster 21 - Inappropriate Affect/Inattention
Cluster 23 - Emotional Expression Blunting
Cluster 26 - General Negative Symptoms

Table 20

Control Scale Items

SSI 06 - Do you ever have fits or difficulty in keeping your balance

SSI 14 - Are you unable to prevent yourself from doing pointless things

SSI 18 - Are you slower recently in everything you do

SSI 22 - Do you sweat very easily, even on cold days

SSI 27 - Are you ever so low in spirits that you sit for hours on end

SSI 37 - Are you a more important person than most people seem to think

SSI 41 - Do you ever lose the use of an arm or leg or face muscle

SSI 43 - Do you suffer from palpitations or breathlessness

SRS 14 - Physical symptoms of anxiety or depression

BPRS 5 - Guilt feelings

Four of the twelve symptom clusters were deleted from the Positive Variable Group. One of these four clusters (Symptom Cluster 25 - SAPS item 33 - "Clanging") was also removed from the Nonparanoid Variable Group. As noted above, the low variation in its scores would have made it difficult to find group differences and is probably the reason for its removal from this group.

The reasons for the removal of the other three clusters are not as clear (Symptom Cluster 10 - "Realization of Strange Thoughts", Symptom Cluster 19 - "Delusions", and Symptom Cluster 22 - "General Hallucinations"). These three clusters do not seem to have any specific content which would explain why they were mathematically separated from the other items. Cluster 10 is in the Nonparanoid Variable Group and Cluster 19 is in the Paranoid Variable Group. Cluster 22 is in neither of these two Variable Groups. Therefore, their relation to the Paranoid-Nonparanoid distinction does not seem an important part of this removal.

Composition of the variable groups. The five different variable groups (paranoid, nonparanoid, positive, negative, and control) will each be discussed in this section. First, the Paranoid Symptom Clusters all appear consistent with the DSM-III-R diagnostic criteria for 295.2x (Paranoid Type; see Appendix G). All but one of these symptom clusters exclusively contain items which directly are related to delusions (e.g., the single item in Paranoid Symptom Cluster 15: SSI 36 "Have you come special power, ability, or influence that is not recognized by other people?"). Paranoid Symptom Cluster 4 contains the only non-delusional symptoms. These symptoms are auditory hallucinations and are combined in the cluster with delusions related to a patient's

thoughts (e.g., SAPS 17 - "Thought Broadcasting"). Thus, these symptom clusters appear very appropriate as indicators of paranoid schizophrenia.

The symptom clusters in the Nonparanoid Variable Group also seem very consistent with the DSM-III-R diagnosis of schizophrenia. These Nonparanoid Symptom Clusters contain several Positive Symptom Clusters that are not related to delusions, including behaviour (Symptom Cluster 17 - "Bizarre Behaviour/Language) and thought disorder (Symptom Cluster 11 - "Sex/Religion Distressing Thoughts"). It also contains several Negative Symptoms Clusters including Symptom Cluster 20 ("Apathy") and Symptom Cluster 21 ("Inappropriate Affect/Inattention"). Thus, the Nonparanoid Variable Group contains many symptoms related to schizophrenia but does not contain either delusions or associated hallucinations.

The items within the Positive Variable Group Symptom Clusters all appear to reflect positive symptoms. It appears to reflect the wide range of positive symptoms, by including items in the Paranoid Variable Group (Symptom Clusters 4, 9, and 15) as well as items in the Nonparanoid Variable Group (Symptom Clusters 17 and 18). The Positive Variable Group also included symptom clusters that were unique to it (Clusters 6, 13, and 24). In general, these three unique symptom clusters did not contain enough information to allow the raters to determine if the clusters reflected paranoid or nonparanoid symptomatology. For example, Positive Symptom Cluster 13 contained only SSI 32: "Do you ever hear voices without knowing where they come from?". A positive response to this question indicates that something is occuring that is beyond the range of "normal" functioning and is

therefore a positive symptom. There is no evidence, however, as to the degree they reflect paranoid symptomatology in that they are "frequent hallucinations related to a single theme" (DSM-III-R, p. 197). Therefore, judgements could not be made about whether such symptom clusters reflected paranoid or nonparanoid symptoms. As a result, these symptom clusters were not included with either group by the raters. Taken together, the eight Positive Symptom Clusters were viewed as allowing for sufficient breadth in coverage to have the Positive Variable Group be a valid reflection of the wide scope of positive symptomatology.

The breadth of the Negative Variable Group is less than that found with the Positive Variable Group. This lessening of breadth is evident in the far fewer number of Negative Symptom Clusters (4) compared to the number of Positive Symptom Clusters (8). Nonetheless, the symptomatology covered by the Negative Symptom Clusters was not overly restricted. They cover a wide range of symptoms including flat affect, avolition, alogia, and anhedonia. These are also the four areas of negative symptoms that are being discussed for inclusion in the DSM-IV (Task Force on DSM-IV, 1991). Thus, while fewer in number, they contain a wide enough variety of symptoms that the resultant Negative Variable Group can be viewed as appropriate for a discussion of negative symptoms.

After the consistency tests, only 10 (or less than half) of the 21 original Control Variable Group items remained. A review of these items indicated that the majority (8 out of 10) are related to physical functioning at some level. Also, the majority (8 out of 10) are SSI items. One of the other two surviving items (BPRS 5 - "Guilt

Feelings") is also based on the patient's subjective reports and not on the rater's observations of the patient's behaviour. All items are related to one of these two themes. Thus, the general theme that runs through these data may be that of the degree of self-report of complaints of physical functioning. It therefore appears that there may be a pattern in these Control Variable Group items that could define a nonschizophrenia symptomatology scale.

Maximum Covariance Analysis

For the sake of illustration of this method of taxometric analysis, the example will continue to be the positive symptom group of eight subscales (i.e., symptom clusters). The first step of the MAXCOV analysis was for two of the subscales (e.g, Positive Symptom Clusters 4 and 6) to be separated from the six remaining subscales (e.g., Positive Symptom Clusters 9, 13, 15, 17, 18, and 24). Second, a seven-point scale was then formed by aggregating the six remaining subscales. This new seven-point scale had scores ranging from 0 to 6.

In the third step, seven different subsamples were formed. Each subsample was comprised of subjects who scored at one level of the seven-point scale created in the second step. For example, the individuals who scored 0 formed one subsample, those individuals who scored 1 formed a second subsample, those individuals who scored 2 formed a third subsample, etc.

In the fourth step, the covariance between the two subscales originally removed (e.g., Positive Symptom Clusters 4 and 6 in step one) would be calculated separately for each of the seven subsamples (e.g., covariance = -0.0221 for the subjects who scored 2). This four-step technique was then replicated until all 28 possible

combinations of "two removed" subsamples and "six remaining" subsamples were examined.

In the fifth step, a mean covariance was calculated at each of the seven levels of subsamples (0 to 6) to reduce sampling variation (e.g., mean covariance = 0.0808 for subjects who scored 2). In the sixth step, to further reduce such sampling variation, Tukey's (1977) "3RH Twice" method of smoothing was employed on these covariance aggregates (e.g., smoothed mean covariance = 0.0122 for subjects who scored 2). The resultant data were then plotted on a graph in the seventh step (both smoothed and unsmoothed data) to determine if the mean level of covariance between two items varied across the corresponding seven point scales. As described in Chapter 1, if two classes do exist in a variable group, then the covariance between two symptom clusters would be a function of the relative proportions of the two classes (e.g., positive and non-positive). If there is a "peak" in the graph, then it is assumed that a class variable exists. If there is no such peak evident in the graph, then no class variable is assumed.

In the present investigation, MAXCOV analyses were undertaken for each of the five variable groups and their raw and smoothed data were plotted: (i) the paranoid symptom scales (see Figure 9), (ii) the nonparanoid symptom scales (see Figure 11), (iii) the positive symptom scales (see Figure 12), (iv) the negative symptom scales (see Figure 13), and (v) the control items (see Figure 14). To allow for an examination across variable groups, all curves were placed on graphs of the same size. Although some of these curves, most notably from the negative symptom scale, were small compared to the overall graph size,

FIGURE 10

Covariance curves based on the average covariances across all 15 pairings of paranoid symptom clusters for each interval on the corresponding 5-point paranoid scale for both raw and smoothed data (n = 100)

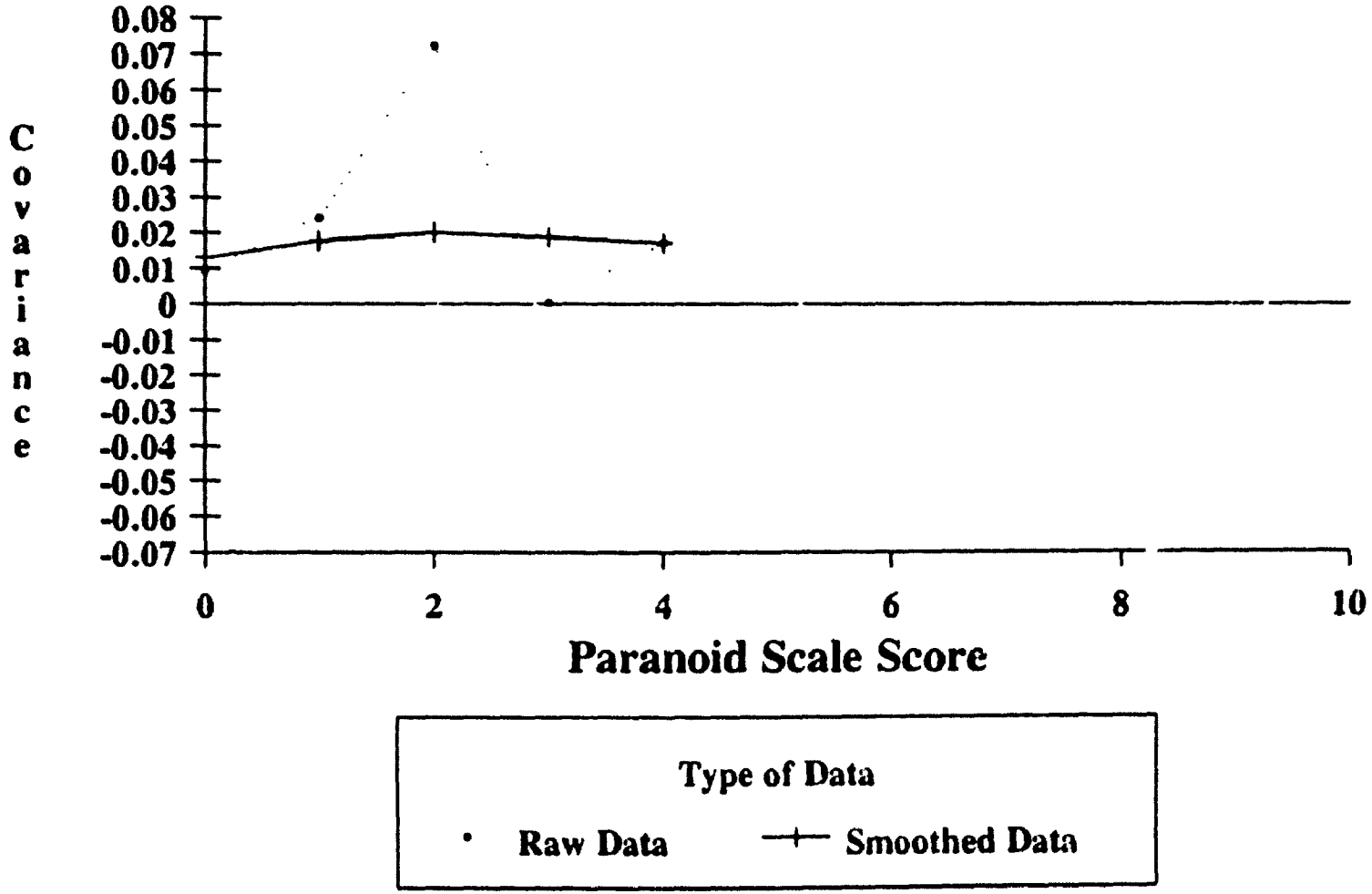

Covariance
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
-0.01
-0.02
-0.03
-0.04
-0.05
-0.06
-0.07
0
2
4
6
8
10
Paranoid Scale Score
Type of Data
Raw Data
Smoothed Data

FIGURE 11

Covariance curves based on the average covariances across all 28 pairings of nonparanoid symptom clusters for each interval on the corresponding 7-point nonparanoid scale for both raw and smoothed data (n = 100)

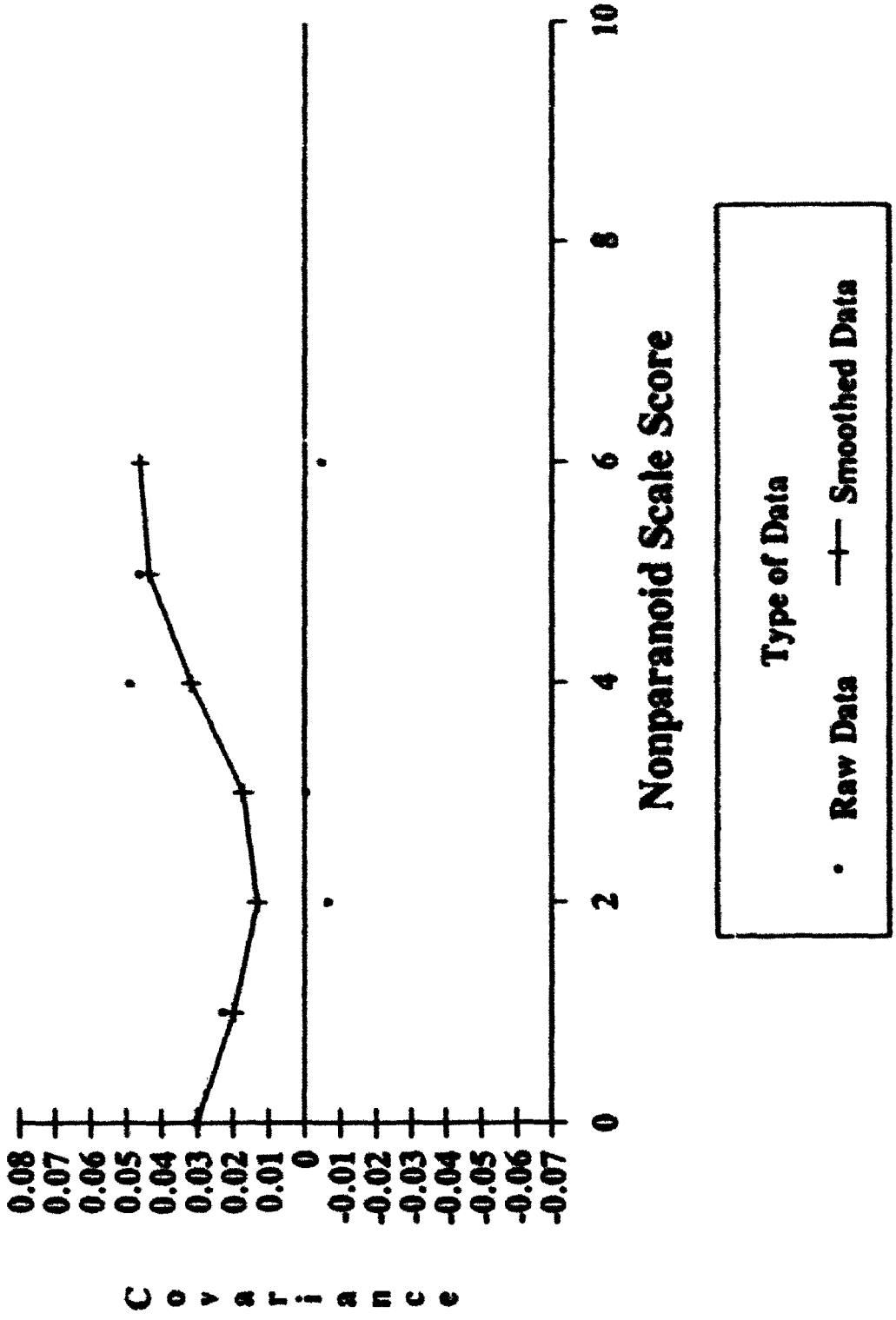

Covariance
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
-0.01
-0.02
-0.03
-0.04
-0.05
-0.06
-0.07
0
2
4
6
8
10
Nonparanoid Scale Score
Type of Data
Raw Data
Smoothed Data

FIGURE 12

Covariance curves based on the average covariances across all 28 pairings of positive symptom clusters for each interval on the corresponding 7-point positive scale for both raw and smoothed data (n = 100)

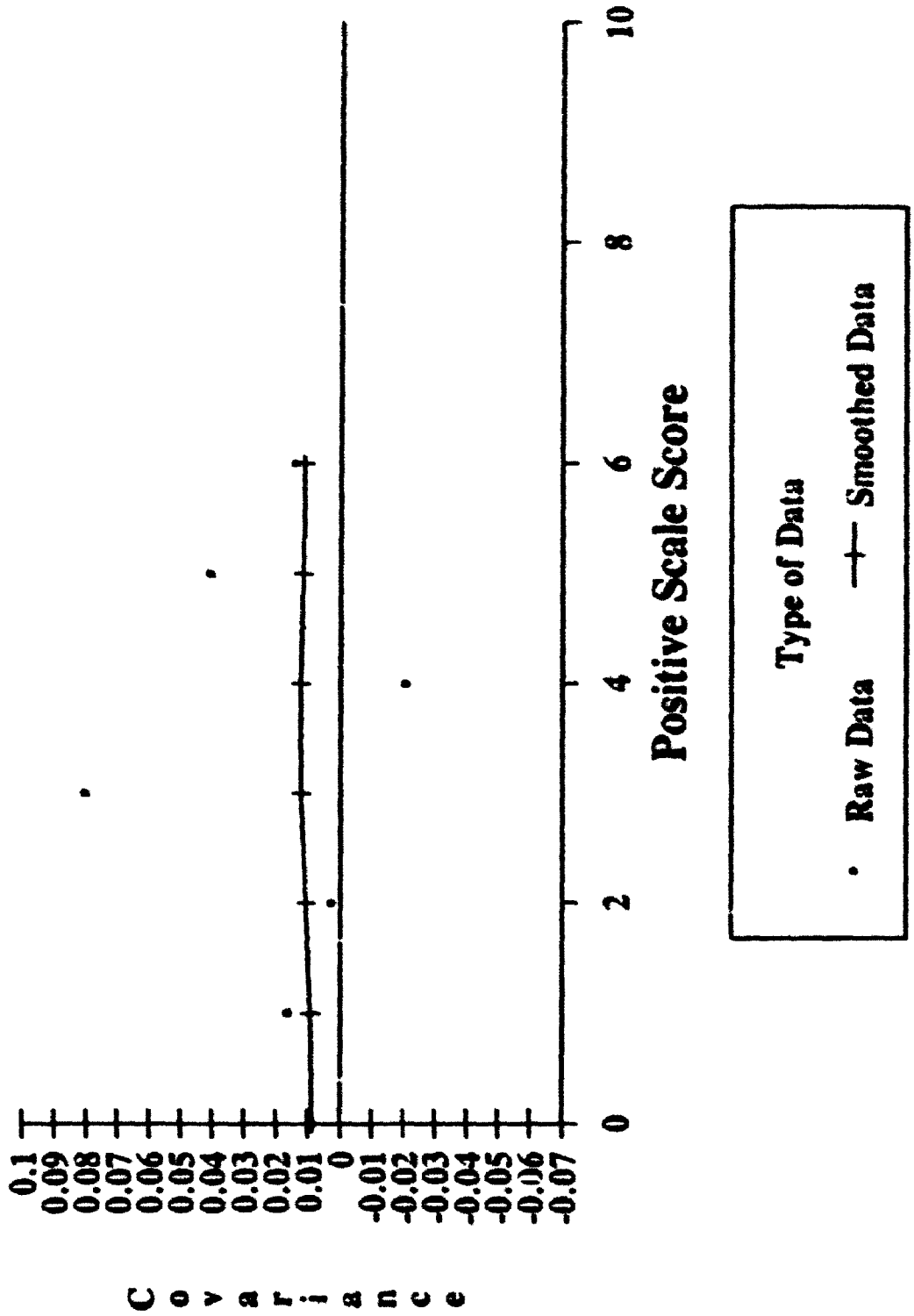

Covariance
0.1
0.09
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
-0.01
-0.02
-0.03
-0.04
-0.05
-0.06
-0.07
0
2
4
6
8
10
Positive Scale Score
Type of Data
Raw Data
Smoothed Data

FIGURE 13

Covariance curves based on the average covariances
across all 6 pairings of negative symptom clusters
for each interval on the
corresponding 3-point negative scale
for both raw and smoothed data (n = 100)

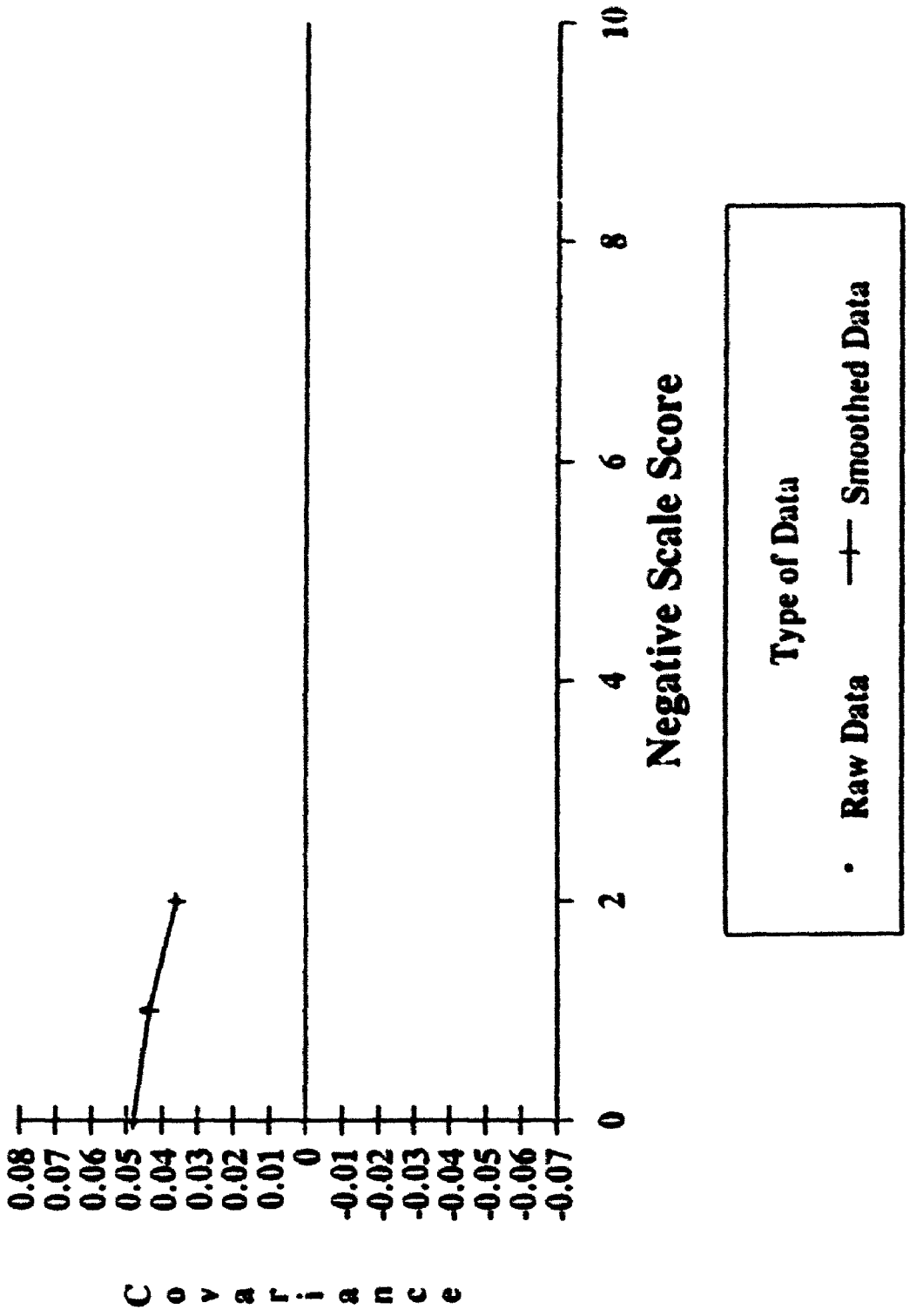
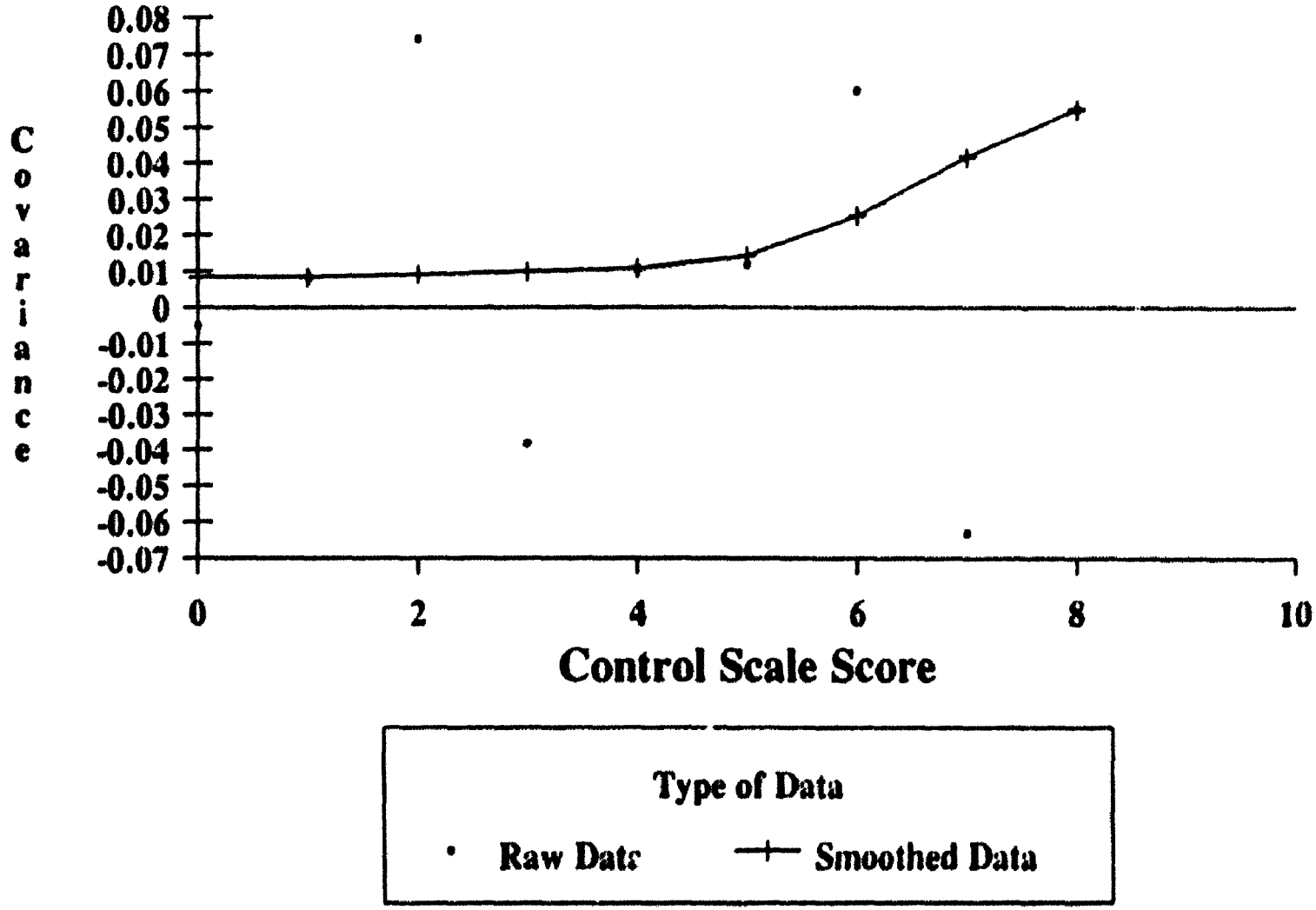

Covariance
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
-0.01
-0.02
-0.03
-0.04
-0.05
-0.06
-0.07
0
2
4
6
8
10
Negative Scale Score
Type of Data
Raw Data
Smoothed Data

FIGURE 14

Covariance curves based on the average covariances
across all 45 pairings of control items
for each interval on the
corresponding 9-point control scale
for both raw and smoothed data (n = 100)


Covariance
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
-0.01
-0.02
-0.03
-0.04
-0.05
-0.06
-0.07
0
2
4
6
8
10
Control Scale Score
Type of Data
Raw Data
Smoothed Data

putting them on a smaller graph would have made comparisons to curves plotted on different metrics difficult.

An examination of the smoothed curves from the four schizophrenia variable groups indicates that they do not display "peakedness". While the paranoid symptom scales might initially be thought of as "peaked" prior to smoothing, a comparison to previous MAXCOV research supports even this raw data as being "smooth". For example, in Figure 6 the gender curve ranged from approximately 0.00 to 0.25, more than 200% more peaked than the paranoid raw data. Therefore, these four smoothed curves appeared consistent with the type of curve expected if a dimensional model was appropriate. Thus, the four schizophrenia symptom clusters support each of these four groups of patients as being on a dimension. For example, there do not appear to be two groups of patients in which some are in a "paranoid" class and others are not in the class. This pattern is consistent with the two-factor model in that the patients would be seen as being on a paranoid-nonparanoid continuum.

The fifth variable group, the control items, displays an upward rise at the right end. As noted in the introduction, this type of upward-curving right-hand peak has been related to a class model when the base rate for one of the classes in the sample was less thar 0.25. Thus, it would appear that, whatever this group of items measures, it may be a class variable. The implications for this result, as well as for the other MAXCOV analyses, are discussed in more detail in Chapter 4.

Comparison of Base Rate Estimates

As noted above, the primary hypothesis underlying this method was that different sets of mathematical formulae for estimating base rates,

which were not derivable from one another, should provide consistent results only if there was some form of an underlying dichotomy. Two different methods were employed in these analyses. The first method of estimating base rates, described by Meehl (1973), involves several steps. In the first step, each subject was placed into one of two groups for each variable (i.e., symptom cluster) depending on whether he or she had a score of 0 or 1. For example, with Positive Symptom Cluster 4 there were 2 groups of subjects.

In the second step, the average covariance between unique pairs of the remaining variables was calculated separately for the subjects in each of these two groups. For the present example, this would mean that with Positive Symptom Cluster 4, there was a covariance calculated for each one of the 21 pairs of variables for each of the two groups (i.e., 42 covariance estimates for this variable).

Third, the maximum covariance for each group of variables was viewed as an estimate of 1/4 of a constant (K) which was defined as the fixed product of the latent mean differences. With Positive Symptom Cluster 4, the maximum covariance was 0.1067 (covariance of Positive Symptom Clusters 17 and 24 when the grouping variable was 0). Therefore, $K = .4268$.

Fourth, given that:

$$\text{cov}(\text{(symptom cluster } y\text{) X (symptom cluster } z\text{)}) = p(1-p)K,$$

for each level (0, 1) of the symptom cluster, the quadratic equation:

$$Kpp - Kp + \text{cov}(yz) = 0,$$

gives two solutions for p (the base rate of the underlying class). The higher of the two solutions was employed when the group = 1 (class presence) and the lower solution was for when the group = 0 (class

absence). For the Positive Symptom Cluster 4 example, the covariance of Positive Symptom Clusters 6 and 9 were 0.0565 at group = 0 and 0.0612 at group = 1. The resultant $\underline{p}$ for each were group was 0.1570 at group = 0 and 0.9090 at group = 1.

Fifth, for each separate covariance, a proportionality estimate was calculated by multiplying the $\underline{p}$ by the number of subjects in the group. To continue this Positive Symptom Cluster 4 example, the (6, 9) covariance would give proportionality estimates of 8.0 for group = 0 and 44.5 for group = 1. Sixth, these estimates were then averaged within a group. For Positive Symptom Cluster 4, the mean group estimates were 5.2 for group = 0 and 44.2 for group = 1. Finally, the two group (0,1) means were totalled and this sum was the first estimate of the base rate of the symptom cluster. With Positive Symptom Cluster 4, this first base rate estimate was 49 out of 100 subjects, or 0.49.

The second base rate estimate technique (Golden & Meehl, 1979) was based, in part, on the result of consistency test 3 (see above). In that test, each subject had a separate scale score calculated for each variable (i.e., symptom cluster) in a variable group. This score was calculated by adding together the scores on that group's remaining variables. For Positive Symptom Cluster 4, the scores on the remaining seven symptom clusters were totalled. The subjects were then ranked according to this variable's scale score. The top quartile of subjects in this variable's scale score ranking were seen as the best estimate of that variable group and the bottom quartile were seen as the best estimate of the alternate group. Therefore, in this example, Positive Symptom Cluster 4 scale's top quartile of subjects were seen as its best representatives of positive subjects ($\underline{p}$(Pos)) while the bottom quartile

represented non-positive subjects ($\underline{p}$(Non)). The average scores on Positive Symptom Cluster 4 were then calculated separately for the top quartile (0.86) and bottom quartile (0.13) groups.

Golden and Meehl (1979) described a base rate estimate for the underlying class as being calculable from these data. First, they developed the formula for the overall mean of the variable:

$$\underline{P}(x\underline{p}(Pos)) + \underline{Q}(x\underline{p}(Non)) = \underline{x},$$

where $\underline{P}$ was the base rate estimate for the Positive Variable Group,

$\underline{Q} = 1 - \underline{P}$, and

$\underline{x}$ was the mean variable (Positive Symptom Cluster) score.

The formula was then rearranged to develop a formula to estimate the base rate $\underline{P}$:

$$\underline{P} = (\underline{x} - x\underline{p}(Non)) / (x\underline{p}(Pos) - x\underline{p}(Non)).$$

For Positive Symptom Cluster 4, $x\underline{p}(Pos) = 0.86$, $x\underline{p}(Non) = 0.13$, and $\underline{x} = 0.49$. Putting these values into the above equation, the second base rate estimate ($\underline{P}$) from Positive Symptom Cluster 4 is 0.49.

The base rate estimates from these two techniques are listed in Table 21 (Paranoid and Nonparanoid), Table 22 (Positive and Negative), and Table 23 (Control). When Gangestad and Snyder (1985) reviewed the base rate estimates from their results, they only listed such a table for the eight items related to the variable that peaked in their MAXCOV analyses. The two sets of estimates were not compared statistically. Instead, they were discussed in terms of their inter-estimate mean base rate consistency. Gangestad and Snyder, in their review of the base rate estimates on the variables which did not display peaked MAXCOV curves, only discussed these overall mean estimates. They did not

Table 21

Paranoid/Nonparanoid Base Rate Estimates

	Cluster Number	Base Rate 1	Base Rate 2
Paranoid	2	36	45
	4	48	46
	9	47	47
	15	38	17
	16	53	58
	19	48	49
Nonparanoid	10	45	47
	11	36	40
	14	35	38
	17	49	50
	18	44	28
	20	43	27
	21	48	39
	26	48	35

Snyder for the latent class variable example. These results were also not dissimilar to what Gangestad and Snyder described for the latent continuum variable example. The present study (with a sample size of 100) may have had more variance in its estimates than did Gangestad and Snyder (with a sample of 1918 undergraduates). Because the technique of comparing base rate estimates has not been widely reapplied, there is no data to examine the degree that sample size affects the various base rate calculations and the subsequent comparisons.

Further Insight Into the Two-Factor Model of Schizophrenia Symptomalogical Classification

Support for a dimensional structure for these four schizophrenia variable groups is available both from the MAXCOV analyses and from the comparisons of base rate estimates. Because the MAXCOV analyses have been more widely employed in previous research and have been the subject of numerous Monte Carlo studies, these results are clearer and easier to interpret. Therefore, the present discussion will focus on them to the exclusion of the comparisons of base rate estimates. Nothing in the results of the base rate estimate comparisons, however, would disagree with any of the following points.

When the content of the four Schizophrenia Variable Groups is analyzed, the amount of overlap between them becomes evident. First, the eight Positive Symptom Clusters contain both three Paranoid Symptom Clusters and two Nonparanoid Symptom Clusters. It also contains three symptom clusters that are not placed into either group. Of these three symptom clusters, one rater listed one of them as Paranoid while a second rater listed another of the symptom clusters as Nonparanoid,

Table 23

Control Base Rate Estimates

Item Number	Base Rate 1	Base Rate 2
2	39	64
4	33	36
5	61	39
6	38	30
7	36	61
8	45	52
9	26	52
10	41	44
16	34	60
19	47	35

review the individual item estimates as they did with the other variables they studied.

An examination of the present data indicates that some symptom clusters make similar predictions for the two base rates. As can be seen in Table 22, for example, Positive Symptom Cluster 4 each base rate had an estimate of 49. Even within the same variable group, however, other symptom clusters had widely divergent base rate estimates. For example, Positive Symptom Cluster 18 had estimates of 48 and 40. Positive Symptom Cluster 17, on the other hand, had estimates which were as disparate but in the opposite direction (estimate 1 = 49; estimate 2 = 57). Gangestad and Snyder's (1985) largest items range for their discontinuous variable was estimate 1 = 40 and estimate 2 = 49.

The ranges were also much wider than that described by Gangestad and Snyder across the set of eight estimates of the discontinuous variable. Their first base rate estimates only ranged from 0.36 to 0.44 and the second set of estimates ranged from 0.39 to 0.49. In this investigation, two groups of variables had eight symptom clusters: Nonparanoid and Positive. For Base Rate Estimate 1, the Nonparanoid range was 33 to 49 while the Positive range was 29 to 49. Base Rate Estimate 2 had a Nonparanoid range of 27 to 50 and a Positive range of 33 to 57.

Because of the very small sample sizes of symptom clusters in some of these variable groups, most notably the Negative Symptom Clusters (n = 4), many statistics for comparing these sets of estimates were seen as inappropriate. For example, correlated *t*-tests would have had very little power to note anything but the grossest of mean differences. Measures of similarity between the two sets in the patterns of their

estimates were also limited. Nonetheless, nonparametric correlations, such as Kendell's Tau were possible (see Table 24). After a Bonferroni correction for the number of Kendell's Tau calculated, none of these sets of base rate estimates were correlated significantly.

These findings are not consistent with the systematic operation of data structure specified by the taxon/class model. Thus, as was evident in the review of the MAXCOV curves, the four schizophrenia symptom clusters support each of these four groups of patients as being on a dimension. This pattern is consistent with the two-factor model in that the patients would be seen as being on a continuum (see below).

Table 24

Correlations of Base Rate Estimates

Group(Size)	Kendell's Tau
Paranoid (6)	0.40
NonParanoid (8)	0.36
Positive (8)	0.57
Negative (4)	0.00
Control (10)	-0.16

Note: None are significant after a Bonferroni correction

CHAPTER 4 - DISCUSSION

Maximum Covariance Analysis

Preliminary discussion. Before discussing the implications of the present results, two general points should be made concerning the MAXCOV results. First, it should be noted some of the raw average covariances were negative. These covariances can be seen in Figure 11 (at one of seven points), Figure 12 (at three of seven points), and Figure 13 (at three of nine points). It was not until the curve was smoothed that all seven of these negative average covariances became positive. This pattern has been reported in previous MAXCOV research. For example, Lenzenweger and Korfine's (1992) investigation of schizotypy resulted in an unsmoothed (raw) average covariance that was negative (see Figure 8). After the curve was smoothed, however, that average covariance became positive. Thus, raw negative average covariances do not invalidate the results.

Second, the ranges of the present covariances are consistent with those ranges found in previous research. The four schizophrenia variable groups all had their smoothed average covariances vary somewhere between 0.0087 and 0.0480. Such a range of smoothed average covariances is consistent with that of several other MAXCOV investigations. For example, Strube's (1989) MAXCOV results were graphed and appeared to range from approximately 0.005 to 0.021. Gangestad and Snyder's (1985) smoothed analyses were also graphed and appeared to have a range from 0.010 to 0.042 (see Figure 5). Thus, the results of these MAXCOV analyses were within the range of the average covariances in other studies.

Primary discussion. None of the four schizophrenia variable groups displayed smoothed (average) covariance curves with any of the "peakedness" that would be an indication of a latent dichotomous class variable. Thus, the paranoid, nonparanoid, positive, and negative variable groups all supported an underlying continuum in the data. Previous research indicates that data might divide patients into one of two groups, then a "peaked" curve occurs. For example, Trull et al. (1990) used items such as "no menstruation" and "male first name" to investigate gender with MAXCOV. Their results indicated a strongly "peaked" smoothed (average) covariance curve (see Table 6). Thus, the present results can be interpretted as strong evidence of the lack of presence of a dichotomy among the patients in any one of these four schizophrenia variable groups.

The control items group, however, did display a form of peakedness. Comparing it to previous research employing MAXCOV suggests that the split involved less than 25% of the patients fitting into one of two separate taxons (e.g., see Table 11; Lenzenweger & Korfine, 1992). While it is unclear what the control group items are measuring, it may be the case that they are all related to somatoform disorders which previous research has suggested may be categorical and not on a continuum (Cloninger, Sigvardsson, von Knorring, & Bohman, 1984).

Comparisons of Base Rate Estimates

The results of these analyses were not as well defined as with the MAXCOV analyses. The main reason for this lack of definition was that the method has not been employed beyond Gangestad and Snyder's (1985) initial discussion of the technique. The results of the present investigation were not as similar as what was described by Gangestad and

Snyder for the latent class variable example. These results were also not dissimilar to what Gangestad and Snyder described for the latent continuum variable example. The present study (with a sample size of 100) may have had more variance in its estimates than did Gangestad and Snyder (with a sample of 1918 undergraduates). Because the technique of comparing base rate estimates has not been widely reapplied, there is no data to examine the degree that sample size affects the various base rate calculations and the subsequent comparisons.

Further Insight Into the Two-Factor Model of Schizophrenia

Symptomalogical Classification

Support for a dimensional structure for these four schizophrenia variable groups is available both from the MAXCOV analyses and from the comparisons of base rate estimates. Because the MAXCOV analyses have been more widely employed in previous research and have been the subject of numerous Monte Carlo studies, these results are clearer and easier to interpret. Therefore, the present discussion will focus on them to the exclusion of the comparisons of base rate estimates. Nothing in the results of the base rate estimate comparisons, however, would disagree with any of the following points.

When the content of the four Schizophrenia Variable Groups is analyzed, the amount of overlap between them becomes evident. First, the eight Positive Symptom Clusters contain both three Paranoid Symptom Clusters and two Nonparanoid Symptom Clusters. It also contains three symptom clusters that are not placed into either group. Of these three symptom clusters, one rater listed one of them as Paranoid while a second rater listed another of the symptom clusters as Nonparanoid,

None of the three raters listed the third symptom cluster as being in either the Paranoid or the Nonparanoid Variable Group.

The four Negative Symptom Clusters can be divided into three Nonparanoid Symptom Clusters and one that was not placed in either the paranoid or the nonparanoid group. Prior to the consistency tests, however, it was placed in the Negative Variable Group by two of the three raters. Thus, the Negative Symptom Clusters were generally nonparanoid while the Positive Symptom Clusters were both paranoid and nonparanoid.

The same four sets of Schizophrenia Variable Groups can be viewed from an opposite perspective. The Nonparanoid Variable Group had two symptom clusters that were also in the Positive Variable Group, three symptom clusters in the Negative Variable Group, and three that were in neither group. Prior to the consistency tests, one of these three symptoms was rated by all three raters as being a Positive Symptom Cluster. It failed to pass the Positive Symptom consistency tests, however, and was not included in the final group of eight symptom clusters.

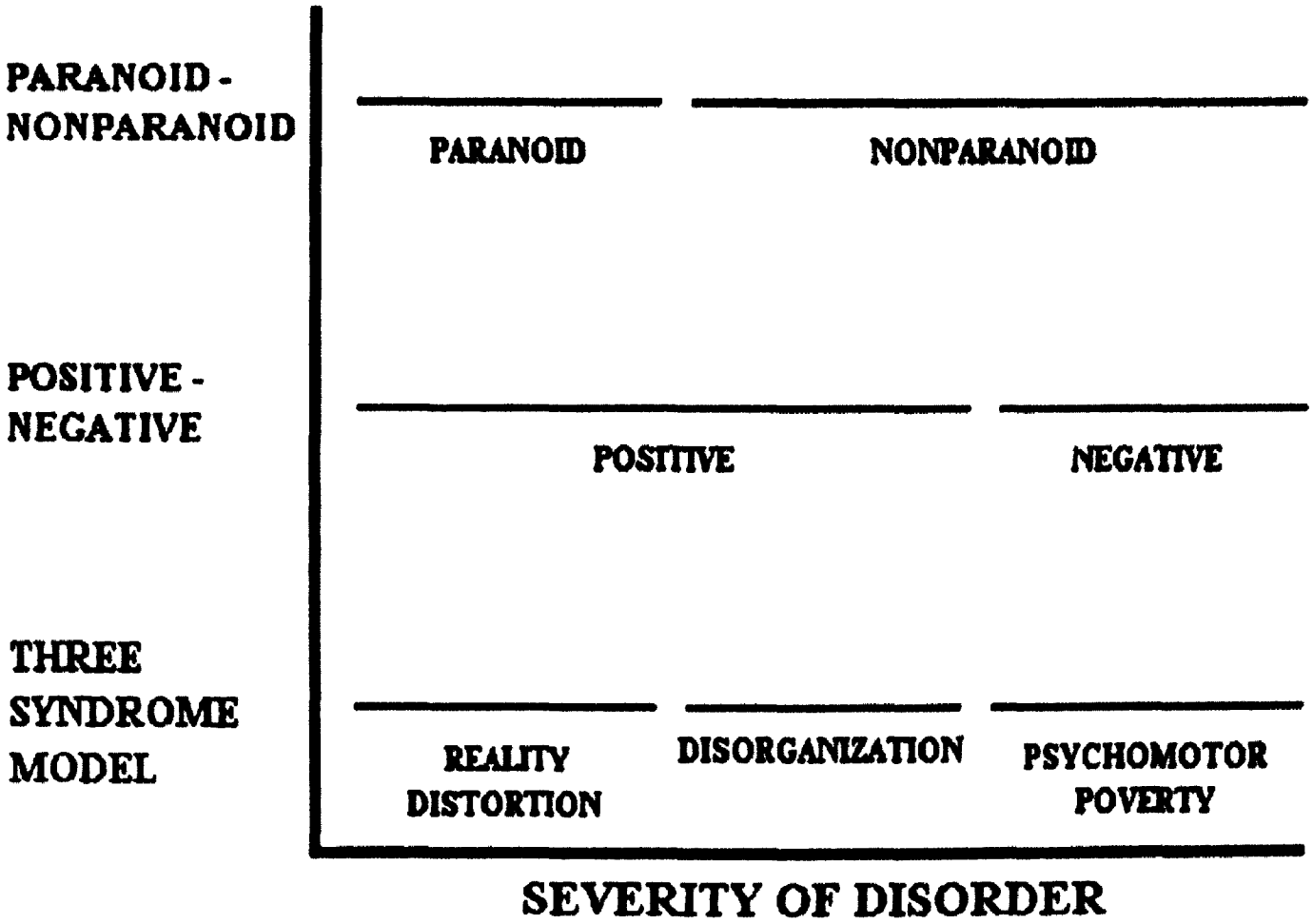
Three of the six Paranoid Variable Group Symptom Clusters were also rated as Positive Variable Group Symptom Clusters. The three remaining symptom clusters were classified as neither positive nor negative. All three raters had rated one of these nonclassified symptom clusters as positive. It did not pass the Positive Symptom consistency tests, however, and was not included in that variable group. The other two Paranoid Symptom Clusters were rated as positive by only one of the three raters but had no ratings as being negative.

When these two opposite perspectives are taken together, a graph similar to the one found in Figure 15 can be developed. In this graph, the two-factor model's proposed severity of disorder continuum related to paranoid and nonparanoid schizophrenia can be conceptualized. The Nonparanoid Symptom Clusters would cover a wider range of symptoms. More variation within this Variable Group would lead to greater variability between the nonparanoid scales. As a result, the Nonparanoid Variable Group Symptom Clusters have less consistency. This difference between paranoid and nonparanoid consistencies was discussed earlier with regard to the finding that the nonparanoid measures tended to have lower levels of interscale correlations. In Figure 15, the space between the two groups of symptom clusters would include symptoms that could not be classified as either paranoid or nonparanoid and does not represent an actual break between the two variable groups.

The positive-negative dichotomy in the symptomatology of schizophrenia can also be conceptualized from the perspective of the two-factor model's severity of disorder continuum. In this instance, the Negative Variable Group has a narrower range of symptom clusters than does the Positive Variable Group. Andreasen's original research on these scales indicated that the SANS had a higher internal consistency than the SAPS (Andreasen & Olsen 1982). This pattern is also consistent with factor analyses which have found one factor on the SANS but more than one factor on the SAPS (e.g., Arndt, Alliger, & Andreasen, 1991; Kulhara, Kota, & Joseph, 1986). Also, Negative Symptom Clusters are related only to Nonparanoid Symptom Clusters while the Positive Symptom Clusters are related to both Paranoid and Nonparanoid Symptom Clusters. Various studies have supported the view that the paranoid

FIGURE 15

Conceptualization of three previous models of the symptomatology of schizophrenia as they might relate to the two-factor model's severity of disorder continuum (breaks within a line represent an area where symptoms are not clearly defined as belonging to one do not represent an area where patients do not exist)


PARANOID - NONPARANOID
PARANOID
NONPARANOID
POSITIVE - NEGATIVE
POSITIVE
NEGATIVE
THREE SYNDROME MODEL
REALITY DISTORTION
DISORGANIZATION
PSYCHOMOTOR POVERTY
SEVERITY OF DISORDER

symptoms of prominent delusions (often associated with thematic hallucinations) form a cohesive group that is separable from other positive symptoms (Neufeld & Williamson, in press). In addition, Paranoid Symptom Clusters are seen as only related to Positive Symptom Clusters while Nonparanoid Symptom Clusters are related to both Positive and Negative Symptom Clusters.

When a graph is drawn to plot these aspects of the Positive and Negative Variable Groups on the two-factor model severity of disorder continuum, then a figure similar to that found in Figure 15 emerges. Note that, as with the paranoid-nonparanoid portion of the graph, the space between the positive and negative symptoms does not represent a break between the two variable groups. Instead, it represents the often-noted problem in defining symptoms at their border (Neufeld & Williamson, in press; Sommers, 1985).

"Negative" schizophrenia would then be seen as the more severe of the two forms and one that could develop with chronicity. Many of the same general differences found between paranoid and nonparanoid schizophrenics can also be found between positive and negative schizophrenics. In their review of the literature, McGlashan and Fenton (1992) found that evidence exists that negative symptoms are more associated with poorer premorbid social functioning, are related to poorer premorbid work adjustment, are perhaps present with a greater family history of psychotic disorders, evidence greater twin concordance in symptoms, dominate the symptomatology of chronic schizophrenia, are more often associated with poor long-term outcome, and may be related to an earlier age of onset. To account for the relations of the two types of symptoms, Fenton and McGlashan (1992) proposed a "unidimensional

bipolar" model of schizophrenia. It proposes "positive and negative subtypes as existing at opposite ends of a single bipolar continuum" (p. 183). They proposed this model to allow for some co-existence of these two subtypes in the same individual as they would be "semi-independent". While such research and theory are in their infancy compared to paranoid-nonparanoid differentiae research, such initial findings are consistent with the relations depicted in Figure 15.

It would be appropriate at this stage to discuss how three syndrome models of schizophrenic symptomatology relate to the severity of disorder continuum, the paranoid-nonparanoid variable groups, and the positive-negative variable groups (as described in Chapter 1). Researchers such as Liddle (1987) and Arndt et al. (1991) have performed factor analyses on symptom groups and found three general syndromes. Liddle described these syndromes as: (1) psychomotor poverty syndrome - dominated by negative symptoms, (2) disorganisation syndrome - dominated by nonparanoid positive symptoms such as incoherence of speech, and (3) reality distortion syndrome - dominated by the paranoid symptoms of delusions and hallucinations. As can be seen in Figure 15, these three groups of symptoms can be placed on the severity of disorder continuum in a manner consistent with their relations with the other two sets of variable groups.

It is interesting to note, however, that while the symptoms of schizophrenia may cluster into three distinct groups, this does not mean that the patients cluster in the same manner. Liddle and Barnes (1990) attempted to classify 57 schizophrenic patients into three separable groups according to their symptom-factor scores. Of these 57 patients, 35 exhibited "definite evidence of more than one syndrome" (p. 560).

Liddle and Barnes also reported that "scatter plots of factor scores reveal no evidence that the patients segregate into identifiable groups" (p. 560). Thus, while the symptoms may fall into three distinguishable groups of symptoms, that does not mean that the patients fall into three easily separable groups.

It is sometimes difficult to clearly picture how three separable sets of different symptoms may be related to different levels of one single disorder. An analogy may be drawn to human pregnancy to show how such changes in symptoms are possible. While not a "disorder", pregnancy is a physiological state that does have certain clusters of symptoms associated with different stages. In the first trimester, a woman may notice such symptoms as nausea, dizziness, and increased urinary frequency. In the second trimester, these symptoms may disappear and be supplanted with weight gain and an increase in energy. In the third trimester, the weight gain will continue but a woman may lose her burst of energy. She may experience difficulty in sleeping, leaking colostrum, Braxton Hicks contractions, increased urinary frequency, and concentration problems. A naive researcher could conclude, based on symptomatology alone, that the three different groups of symptoms represent three different groups of women with three different "disorders". It is evident, however, that even though different clusters of symptoms are associated with different stages of pregnancy, this course is not the same as defining different disorders.

Schizophrenia can also be seen as a disorder which can progress over time. Organized psychotic features, such as delusions of grandeur, may be evident initially. As the disorder progresses, this organization fails, and the symptom picture becomes dominated by other "new"

(i.e., positive) symptoms reflecting the growing disorganization (e.g., inappropriate affect). In the more severe and final stage of the disorder, the patient gradually loses functioning skills (even positive psychotic symptoms) and develops the negative symptoms (e.g., avolition) consistent with the loss of functioning. Thus, as in pregnancy, the symptom clusters change as the "disorder" progresses but this change does not necessarily indicate that different disorders are present.

It should also be noted that, unlike pregnancy, schizophrenia may also start at one of the latter stages. For example, a patient may initially develop disorganized, yet positive, symptoms. Such individuals would be less "protected" against the disease (e.g., initial onset of the disorder would be at an earlier age such as mid-teens). These individuals, however, would still display a tendency to lose functioning skills and develop negative symptoms.

A general comment should be made at this point as to how the paranoid-nonparanoid and positive-negative variable groups support the continuum of severity of disorder. Since there is both, as discussed above, a large overlap between the two previously-hypothesized divisions, and the four variable groups all had MAXCOV results consistent with latent dimensional models, then the two pairs of variable groups could possibly be linked. If such a combination occured to the groups as presented in Figure 15, then a single dimension of disorder would emerge. At different levels of the dimension, different symptoms would be present (as different symptoms are present at different stages of pregnancy). Thus, the MAXCOV results of the present study offer some initial support for a general factor of schizophrenic

symptomatology similar to the one presented in the present two-factor model.

Directions for Future Research

The results of the present investigation have significant implications for several types of research into schizophrenia. These results strongly suggest that schizophrenia essentially is a unified construct. Thus, the Kraepelian view of schizophrenia as a collection of syndromes with some common symptomatology is not supported. Instead, the lack of discontinuities in this investigation support the Bleulerian perspective of schizophrenia as a single disorder with potentially distinguishable manifestations.

Some schizophrenia researchers have argued that the results of schizophrenia studies must be viewed from the perspective of different subtypes to fully understand the data (e.g., Nasrallah, Olson, Coffman, & Schwarzkopf, 1990; Ramirez & Opler, 1990). In reaction to these arguments, Weinberger, Suddath, Torrey, Christison, and Casanova (1990) wrote: "while this subtyping may have descriptive and occasionally prognostic value and may also rescue research data that are otherwise not significant, the implicit assumption that the illness is etiologically or pathophysiologically heterogeneous is pure speculation" (p. 548). The current results support the position taken by Weinberger et al. (1990).

Thus, future research into various aspects of the disorder need not attempt to look for different processes for different subtypes of patients. Some lines of research, such as the search for evidence of a single gene locus for schizophrenia (e.g., Holzman et al., 1988; Meehl, 1992), are supported by this lack of support for a latent taxon in the

disorder. Also, the previous evidence for a single gene locus from these previous studies is consistent with the current findings. Thus, the two lines of research offer support for one another.

While there was no support for different processes, it does not mean that patients with different clinical manifestations of the disorder do not differ in other characteristics. The wide range of differences enumerated for the paranoid and nonparanoid schizophrenics, as well as the positive and negative schizophrenics, are strong evidence that such differences do exist in a variety of areas (e.g., social competence, neuroanatomy). Nonetheless, such differences can be present even when there is only a single latent entity (cf. the previous description of different symptoms at different stages of pregnancy).

Future research specifically aimed at further investigating the two-factor model for the symptomatology of schizophrenia could develop this current project in several possible directions. One avenue for research could involve an investigation as to how the three syndrome model of schizophrenic symptomatology (Liddle, 1987) relates to the current proposed two-factor model. According to the three syndrome model, the three groups of symptoms are relatively equally distributed. In other words, there is nothing in the literature to indicate that patients should be more likely to score high on any particular combination of syndromes relative to any other possible combination. Thus, the three syndrome model would predict approximately equal representation in the resultant three mixed-pairs groups proposed by DSM-IV ("reality distortion/disorganized", "reality distortion/psychomotor poverty", and "disorganized/psychomotor poverty").

As can be seen in Figure 15, the current two-factor model would predict that the probability of belonging to the "reality distortion/psychomotor poverty" group would be significantly lower than that for the other two mixed-pair groups. Figure 15 could be interpreted as indicating that there should be no mixture of the two groups. It is possible, however, that affective symptoms can contaminate measures of negative symptomatology (Newcomer, Faustman, Yeh, & Csernansky, 1990) especially in acute schizophrenia (Liddle, personal communication, November 6, 1992). Thus, there is a rationale to explain the presence of some patients in the "reality distortion/psychomotor poverty" group in that the patients with reality distortion might also display affective symptomatology. This proposal of different probabilities of belonging to different mixed groups could be tested by performing a factor analysis of SANS-SAPS items, extracting the three syndrome factors, classifying patients, and comparing the levels of membership in the three mixed groups.

After these data have been collected, another possible method of examining the two-factor model could be explored. It is being hypothesized that a single, higher-order factor (severity of disorder) underlies the three syndromes of schizophrenic symptomatology. A higher-order factor analysis could be performed on the results of the prior factor analysis of the SANS-SAPS items (Gorsuch, 1974). This earlier factor analysis would need to be oblique (i.e., allow for correlated factors) and such analyses have been employed in the previous three syndrome research (e.g., Liddle & Barnes, 1990). This investigation would add further support for a more general factor actually underlying the three syndrome model. Such a general factor

would be further evidence consistent with the severity of disorder continuum.

Implications for Clinical Practice

As might be expected, the primary implication for clinical practice is in diagnostics. The present study does not support the subtyping of schizophrenia as mirroring distinct entities, either by the traditional method or by employing the proposed subtypes based on the three syndrome model (Task Force on DSM-IV, 1991). As outlined in the introduction, such categorization may have certain advantages, such as allowing for the easier transmission of information (Frances, 1982). Nonetheless, the clinician employing the category should not lose sight of the reality of the underlying dimension. For example, while a woman may be described as being in her last trimester of her pregnancy, a clinician would recognize that this is a categorization of a nine-month continuum. Also, a patient may be described as having an elevated blood pressure. Nevertheless, the clinician would recognize that blood pressure is on a continuum and would also want to know an exact level (e.g., 180/130 mm Hg). Thus, the division of continua into categories is recognized as appropriate in other areas of medicine.

This recognition needs to spread to include other psychiatric disorders. A study by Lang (1978), which compared the replicability of categorical and dimensional models of diagnostic classification, illustrates the utility of such an inclusion. The first method Lang investigated was a traditional categorization of three groups of psychiatric patients: schizophrenics, neurotics, and alcoholics. The data for the analyses were composed of scores from WAIS subtests, a set of self-concept scales, the scales of the Eysenck Personality

Inventory (EPI), nonstandard empirically-derived MMPI scales, and scales derived from the nurses' observations of the patient. A discriminant function analysis was then undertaken which employed these variables and it correctly classified 110 of the 157 patients. These same patients were then divided into three new subgroups based on scores of the Neuroticism scale of the EPI: low, moderate, and high. Employing the same techniques with the same variables, the number of correctly classified increased to 142 of the 157 patients. Lang concluded that this increase in accuracy gave some provisional support for employing dimensional criteria over categorical criteria in classification. While the inclusion of such dimensional approaches is probably not possible in the DSM-IV (Frances, First et al., 1991), the initiation of a "Task Force on DSM-V" may begin shortly after publication of the DSM-IV (Zimmerman, 1988). Whenever the task force begins to review the DSM-IV, this increasing recognition of the importance of dimensions in understanding and diagnosing mental disorders will have to be addressed.

Summary

The current investigation examined the severity of disorder factor of the proposed two-factor model of schizophrenic symptomatology. A semi-structured interview was administered to one hundred patients and a series of symptomatology scales were submitted to ratings. Initial analysis indicated that, in general, the results were both reliable and consistent with previous research in schizophrenia.

The item-level data were clustered to reduce redundancies in the large amount of data (95 schizophrenia variables) and 26 symptom clusters resulted. These symptom clusters were then rated and subsequently placed into four schizophrenia variable groups (paranoid,

nonparanoid, positive, and negative). The four variable groups were put through a series of consistency tests to determine if their symptom clusters were related empirically as well as theoretically. The symptom clusters were then kept in a variable group if they met both types of relations.

Each of these variable groups were analyzed with MAXCOV techniques to determine if they measured a single underlying construct. The resultant graphs were consistent with a dimensional model for the four groups. (A similar set of analyses on a set of control items unrelated to schizophrenia resulted in a graph consistent with a categorical model). There was also some additional support for the dimensional model from comparisons of mathematically-independent categorical-model-based methods of calculating base rate estimates. A further examination of the four variable groups indicated that the results offered initial support for the two-factor model.

The paranoid-nonparanoid and positive-negative divisions offered strong support for the continuum of severity of disorder when they were combined. First, there was a large overlap between the two previously-hypothesized sets of divisions (e.g., positive symptoms could be either paranoid or nonparanoid). Second, the four variable groups all had MAXCOV results consistent with latent dimensional models. If the four groups were linked, as sugggested in Figure 15, then a single severity of disorder factor would result.

These findings have significant implications for future research. First, they offer strong support for a Bleulerian, and not Kraepelian, view of the schizophrenia as a unified whole which has different presentations. As a result, research in several areas need not become

primarily concerned with trying to take into account differences between groups of schizophrenia patients but look for commonalities amongst them (e.g., single-locus gene research). Second, research could also continue with the two-factor model. One potential area of research would be a comparison of this model with the three syndrome model of symptomatology (e.g., Liddle, 1987). The two models are compatable in many respects and their differences are potentially testable. Preliminary discussions currently are underway with other schizophrenia researchers to link symptom data bases to investigate these and other research questions.

These results also have potentially significant implications for clinical practice, particularly in diagnostics. The present study does not support the subtyping of schizophrenia as mirroring actual distinct entities. The division of continua into categories is recognized as appropriate in other areas of medicine. This recognition needs to spread to include the diagnosis of psychiatric disorders so clinicians can retain the familiar classifications but recognize the underlying continuum. The inclusion of such dimensional approaches is probably not possible in the DSM-IV. Nonetheless, the initiation of the "Task Force on DSM-V" may begin shortly after publication of the DSM-IV and the increasing recognition of the importance of dimensions in diagnosing mental disorders will need to be addressed.

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Appendix A

Schneider's First Rank Symptoms

A. Audible thoughts - the patient hears voices speaking his or her thoughts aloud

B. Voices discussing - the patient hears voices discussing or arguing about him or herself

C. Voices describing - the patient hears voices describing his or her activities

D. Delusional percept - the patient experiences a normal perception followed by a delusional interpretation of special significance to that perception

E. Somatic passivity - the patient is a passive and reluctant recipient of bodily sensations imposed from outside of him or her

F. Thought insertion - the patient experiences his or her own thoughts as if they were being imposed from outside of him or her

G. Thought withdrawal - the patient believes that his or her thoughts are being removed due to something or someone from outside of him or her

H. Thought broadcast - the patient believes that his or her thoughts are being transmitted to others

I. Made affect - the patient is made to feel certain affects due to imposition or control from outside of him or her

J. Made impulses - the patient is made to have certain impulses due to imposition or control from outside of him or her

K. Made volitions - the patient is made to have certain motor activities due to imposition or control from outside of him or her

Appendix B

St. Louis (Feighner) Criteria

For a diagnosis of schizophrenia, A through C are required.

A. Both of the following are necessary:
   (1) A chronic illness with at least six months of symptoms prior to the index evaluation without return to the premorbid level of psychosocial adjustment.
   (2) Absence of a period of depressived or manic symptoms sufficient to qualify for affective disorder or probable affective disorder.

B. The patient must have at least one of the following:
   (1) Delusions or hallucinations without significant perplexity or disorientation associated with them.
   (2) Verbal production that makes communication difficult because of lack of logical or understandable organization. (In the presence of muteness the diagnostic decision must be deferred).

C. At least three of the following manifestations must be present for a diagnosis of "definite" schizophrenia and two for a diagnosis of "probable" schizophrenia:
   (1) Single.
   (2) Poor premorbid social adjustment or work history.
   (3) Family history of schizophrenia.
   (4) Absence of alcoholism or drug abuse within one year of onset of psychosis.
   (5) Onset of illness prior to age 40.

Appendix C

New Haven Schizophrenia Index

Checklist of symptoms

1. (a) Delusions (not specified or other than depressive) ____
   (b) Hallucinations (auditory) ____
   (c) Hallucinations (visual) ____
   (d) Hallucinations (other) ____

2. Crazy thinking and/or thought disorder
   Any of the following:
   (a) Bizarre thinking ____
   (b) Autism or grossly unrealistic private thoughts ____
   (c) Looseness of association, illogical thinking, overinclusion ____
   (d) Blocking ____
   (e) Concreteness ____
   (f) Derealization ____
   (g) Depersonalization ____

3. Inappropriate affect ____

4. Confusion ____

5. Paranoid ideation
   (self-referential thinking, suspiciousness) ____

6. Catatonic behaviour
   (a) Excitement ____
   (b) Stupor ____
   (c) Waxy flexibility ____
   (d) Negativism ____
   (e) Mutism ____
   (f) Echolalia ____
   (g) Stereotyped motor acitivity ____

Scoring system

To be considered part of the schizophrenic group, the patient must score on either Item 1 or Item 2a, 2b, 2c and attain a total score of 4 points.

He or she can achieve a maximum of 4 points on Item 1: 2 for the presence of delusions, 2 for hallucinations.

On Item 2 - he or she can score 2 points for any of the symptoms a through c, 1 point for either d through e, and 1 point for each of f and g. He or she can thus score a maximum of 5 points for Item 2.

Items 3, 4, 5, and 6 each receive 1 point.

Note: Where the 4th point is necessary for inclusion in the sample is provided by 2d or 2e, these symptoms are not scored.

Appendix D

12-Point Flexible System

Sign or Symptom	PSE observation or question
A. Restricted affect	Blank, expressionless face. Very little or no emotion shown when delusion or normal material is discussed which would usually bring out emotion.
B. Poor insight	Overall rating of insight.
C Thoughts aloud	"Do you feel your thoughts are being broadcast, transmited, so that everyone knows what you are thinking?" "Do you ever seem to hear your thoughts spoken aloud? (Almost as if someone standing nearby could hear them?)"
D. Waking early (-)	"Have you been waking earlier in the morning and remaining awake?" (Rate positive if 1 to 3 hours earlier than usual.)
E. Poor rapport	Did the interviewer find it possible to establish good rapport with patient during the interview? Other difficulties with rapport.
F. Depressed facies (-)	Facial expression sad, depressed.
G. Elation (-)	Elated, joyous mood.
H. Widespread delusions	How widespread are patient's delusions? How many areas in patient's life are interpretted delusionally?
I. Incoherent speech	Free and spontaneous flow of incoherent speech.
J. Unreliable information	Was the information obtained in this interview credible or not?
K. Bizarre delusions	Are the delusions comprehensible?
L. Nihilistic delusions	"Do you feel that your body is decaying, rotting?" "Do you feel that some part of your body is missing, for example, head, brain, or arms?" "Do you ever have the feeling that you do not exist at all, that you are dead, dissolved?"

Appendix E

Research Diagnostic Criteria for Schizophrenia

There are many different approaches to the diagnosis of schizophrenia. The approach taken here avoids limiting the diagnosis to cases with a chronic or deteriorating course. It includes subjects who would not be considered schizophrenic by many, particularly those subtyped as "acute". However, the criteria are designed to screen out subjects frequently given clinical diagnoses such as: borderline schizophrenia, brief hysterical or situational psychoses and paranoid states. Subjects with a full depressive or manic syndrome which overlaps active psychotic symptoms are excluded and are diagnosed as either Schizo-affective Disorder, Major Depressive Disorder, or Manic Disorder. If the symptoms in A occur only during periods of alcohol or drug use or withdrawal from them, the diagnosis should be Other Psychiatric Disorder because of the likely organic etiology of the symptoms.

A through C are required for the period of illness being considered.

A. During an active phase of the illness (may or may not be present) at least two of the following are required for definite and one for probable:
   (1) Thought broadcasting, insertion, or withdrawal.
   (2) Delusions of being controlled (or influenced), other bizarre delusions, or multiple delusions.
   (3) Somatic, grandiose, religious, nihilistic, or other delusions without persecutory or jealous content lasting at least one week.
   (4) Delusions of any types if accompanied by hallucinatons of any type for at least one week.
   (5) Auditory hallucinations in which either a voice keeps up a running commentary on the subject's behaviors or thoughts as they occur, or two or more voices converse with one another
   (6) Non-affective verbal hallucinations spoken to the subject.
   (7) Hallucinations of any type throughout the day for several days or intermitently for at least one month.
   (8) Definite instances of marked formal thought disorder accompanied by either blunting or inappropriate affect, delusions, or hallucinations of any type, or grossly disorganized behavior.

B. Signs of the illness lasted at least two weeks from the onset of a noticeable change in the subject's usual condition (current signs of the illness may not now meet criterion A and may be residual symptoms only, such as extreme social withdrawal, blunted or inappropriate affect, mild formal thought disorder, or unusual thoughts or perceptual experiences).

C. At no time during the active period (delusions, hallucinations, marked formal thought disorder, bizarre behavior, etc.) of illness being considered did the subject meet the full criteria for either probable or definite manic or depressive syndrome (criteria A and B under Major Depressive or Manic Disorders) to such a degree that it was a prominent part of the illness.

Subtypes of Present Period of Illness

This section is for studies in which there is interest in subtypes of Schizophrenia for the present period of illness.

A. <u>Subtypes based on the course of the present period of Schizophrenia</u>. The following four mutually exclusive categories should be considered for each subject who currently meets the criteria for Probable or Definite Schizophrenia. Note: Some subjects diagnosed initially as Acute may later show a Subacute, Subchronic, or even Chronic course.

(1) Acute Schizophrenia: (a) through (c) are required.
- (a) Sudden onset - less than three months from first signs of increasing psychopathology to any of the core symptoms (criterion <u>A</u>).
- (b) Short course - continuously ill with significant signs of Schizophrenia for less than three months.
- (c) Full recovery from any previous episode.

(2) Subacute Schizophrenia: Course in closer to that of Acute Schizophrenia than that of Chronic Schizophrenia
- Example: First episode with fairly rapid onset and duration of five months.
- Example: Second episode with onset for this episode over a period of six months and full recovery from the first episode.

(3) Subchronic Schizophrenia: Course is closer to that of Chronic Schizophrenia than that of Acute Schizophrenia
- Example: Significant signs of schizophrenia more or less continuously present for at least the past year
- Example: Second period following a previous period in which he did not fully recover.

(4) Chronic Schizophrenia: Significant signs of Schizophrenia more or less continuously present for at least the last two years.

B. Subtypes based on the phenomenology of the present period of schizophrenic illness. The following mutually exclusive subtypes should be considered for each subject who currently meets the criteria for Probable or Definite, to describe the phenomenology of the present condition of the subject. The order here is hierarchical among the first four subtypes.

(1) Paranoid
Throughout the active period of the episode of illness the clinical picture is dominated by the relative persistence of or preoccupation with one or more of the following:
(a) Persecutory delusions.
(b) Grandiose delusions.
(c) Delusions of jealousy.
(d) Hallucinations with a persecutory or grandiose content.

(2) Disorganized [Hebephrenic]
a through c are required.
(a) Marked formal thought disorder.
(b) Either i or ii:
i. Affect which is shallow, incongruous, or silly.
ii. Fragmentary delusions or hallucinations with content not organized into a coherent theme.
(c) Not associated with marked emotional turmoil except during exacerbation.

(3) Catatonic
Throughout the active period of the episode of illness the clinical picture is dominated by any of the following catatonic symptoms:
(a) Catatonic stupor (marked decrease in reactivity to environment and reduction of spontaneous movements and activity).
(b) Catatonic rigidity (maintains a rigid posture against efforts to move him).
(c) Waxy flexibility (maintains postures into which he is placed for at least 15 seconds).
(d) Catatonic excitement (apparently purposelessness and stereotyped excited motor activity not influenced by external stimuli).
(e) Catatonic posturing (voluntary assumption of inappropriate or bizarre posture).

(4) Undifferentiated (or Mixed)
Period of illness meets the criteria for more than one of the previous subtypes or for none of them.

(5) Residual

This category should be used when an individual has had a period of illness in the past that met the criteria A for Schizophrenia but his clinical picture now does not contain any prominent psychotic symptoms, yet residual signs of the illness persist, such as emotional blunting, extreme social withdrawal, eccentric behavior, or mild formal thought disorder. Delusions or hallucinations may be present, but are not prominent.

If a subject meets the criteria for Schizophrenia develops a full depressive syndrome, without an exacerbation of his schizophrenic symptoms, he should receive the additional diagnosis of Depressive Syndrome Superimposed on Residual Schizophrenia. If there is a flare-up of the psychotic symptoms, he should receive the diagnosis of Schizo-affective Disorder, Depressed Type, and the subtyping on the chronicity axis should reflect the duration of the schizophrenic-like symptoms.

(a) through (c) are required for the period of illness being considered.

(a) Once had an active period of illness which met the criteria for Schizophrenia,

(b) The current clinical picture does not contain any prominent psychotic symptoms, although the subject may have some delusions or hallucinations.

(c) Signs of the illness have persisted (e.g., blunted or inappropriate affect, social withdrawal, eccentric behavior, strange or unusual perceptual experiences, mild formal thought disorder) since the time of the active period.

Appendix F

DSM-II Definition of Schizophrenia

This large category includes a group of disorders manifested by characteristic disturbances of thinking, mood, and behavior. Disturbances in thinking are marked by alterations of concept formation which may lead to misinterpretation of reality and sometimes to delusions and hallucinations, which frequently appear psychologically self-protective. Corollary mood changes include ambivalent, constricted, and inappropriate emotional responsiveness and loss of empathy toward others. Behavior may be withdrawn, regressive, and bizarre. The schizophrenias, in which the mental status is attributable primarily to thought disorder, are to be distinguished from the Major affective illnesses which are dominated by a mood disorder. The Paranoid states are distinguished from schizophrenia by the narrowness of their distortions of reality and by the absence of other psychotic symptoms.

Appendix G

DSM-III-R Diagnostic Criteria for Schizophrenia

A. Presence of characteristic psychotic symptoms in the active phase; either (1), (2), or (3) for at least one week (unless the symptoms are successfully treated):

(1) two of the following:
  (a) delusions
  (b) prominent hallucinations (throughout the day for several days or several times a week for several weeks, each hallucinatory experience not being limited to a few brief moments)
  (c) incoherence or marked loosening of speech
  (d) catatonic behavior
  (e) flat or grossly inappropriate affect

(2) bizarre delusions (i.e., involving a phenomenon that the person's culture would regard as totally implausible, e.g., thought broadcasting, being controlled by a dead person)

(3) prominent hallucinations [as defined by (1)(b) above] of a voice with content having no apparent relation to depression or elation, or a voice keeping up a running commentary on the person's behavior thoughts or two or more voices conversing with each other

B. During the course of the disturbance, functioning in such areas as work, social relations, and self-care is markedly below the highest level achieved before the onset of the disturbance (or, when the onset is in childhood or adolescence, failure to achieve expected level of social development)

C. Schizoaffective Disorder and Mood Disorder with Psychotic Features have been ruled out, i.e., if a Major Depressive or Manic Syndrome has ever been present during the active phase of the distubance, the total duration of all episodes of a mood syndrome has been brief relative to the total duration of the active and residual phases of the disturbance.

D. Continuous signs of the disturbance for at least six months. The six-month period must include an active phase (of at least one week, or less if symptoms have been successfully treated) during which there were psychotic symptoms characteristic of Schizophrenia (symptoms in A), with or without a prodromal or redsidual phase, as defined below.

(1) Prodromal phase: A clear deterioration in functioning before the active phase of the disturbance that is not due to a disturbance in mood or to a Psychoactive Substance Use Disorder and that involves at two of the symptoms listed below.

(2) Residual phase: Following the active phase of the disturbance, persistence of at least two of the symptoms noted below, these not being due to a disturbance in mood or to a Psychoactive Substance Use Disorder.

Prodromal or Residual Symptoms:

(a) marked social isolation or withdrawal
(b) marked impairment in role functioning as wage-earner, student, or homemaker
(c) markedly peculiar behavior (e.g., collecting garbage, talking to self in public, hoarding food)
(d) marked impairment in personal hygiene and grooming
(e) blunted or inappropriate affect
(f) digressive, vague, overelaborate, or circumstantial speech, or poverty of speech, or poverty of content of speech
(g) odd beliefs, or magical thinking, influencing behavior and inconsistent with cultural norms, e.g., superstitiousness, belief in clairvoyance, telepathy, "sixth sense", "others can feel my feelings", overvalued ideas, ideas of reference
(h) unusual perceptual experiences, e.g., recurrent illusions, sensing the presence of a force or person not actually present
(i) marked lack of initiative, interests, or energy

Examples: Six months of prodromal symptoms with one week of symptoms from A; no prodromal symptoms with six months of symptoms from A; no prodromal symptoms with one week of symptoms from A and six months of residual symptoms.

E. It cannot be established that an organic factor initiated and maintained the disturbance.

F. If there is a history of Autistic Disorder, the additional diagnosis of Schizophrenia is made only if prominent delusions or hallucinations are also present.

Classification of course. The course of the disturbance is coded in the fifth digit:

1 - Subchronic. The time from the beginning of the disturbance, when the person first began to show signs of the disturbance (including prodromal, active, and residual phases, more or less continuously, is less than two years, but at least six months.

2 - Chronic. Same as above, but with more than two years.

3 - Subchronic with Acute Exacerbation. Reemergance of prominent psychotic symptoms in a person with a subchronic course who has been in the residual phase of the disturbance.

4 - Chronic with Acute Exacerbation. Reemergance of prominent psychotic symptoms in a person with a chronic course who has been in the residual phase of the disturbance.

5 - In Remission. When a person with a history of Schizophrenia is free of all signs of the disturbance (whether or not on medication), "in Remission" should be coded. Differentiating Schizophrenia in Remission from No Mental Disorder requires consideration of overall level of functioning, length of time since the last episode of disturbance, total duration of the disturbance, and whether prophylactic treatment is being given.

0 - Unspecified.

Diagnostic Criteria 295.2x Catatonic Type

A type of Schizophrenia in which the clinical picture is dominated by any of the following:

A. catatonic stupor (marked decrease in reactivity to the environment and/or reduction in spontaneous movements and activity) or mutism

B. catatonic negativism (an apparently motiveless resistance to all instructions or attempts to be moved)

C. catatonic rigidity (maintainance of a rigid posture against efforts to be moved)

D. catatonic excitement (excited motor activity, apparently purposeless and not influenced by external stimuli)

E. catatonic posturing (voluntary assumption of inappropriate or bizarre postures)

Diagnostic Criteria 295.1x Disorganized Type

A type of Schizophrenia in which the following criteria are met:

A. Incohrence, marked loosening of associations, or grossly disorganized behavior.

B. Flat or grossly inappropriate affect.

C. Does not meet the criteria for Catatonic Type.

Diagnostic Criteria 295.3x Paranoid Type

A type of Schizophrenia in which there are:

A. Preoccupation with one or more systemized delusions or with frequent auditory hallucinations related to a single theme.

B. None of the following: incoherence, marked loosening of associations, flat or grossly inappropriate affect, catatonic behavior, grossly disorganized behavior.

Specify stable type If criteria A and B have been met during all past and present active phase of the illness.

Diagnostic Criteria 295.9x Undifferentiated Type

A type of Schizophrenia in which there are:

A. Prominent delusions, hallucinations, incoherence, or grossly disorganized behavior.

B. Does not meet the criteria for Paranoid, Catatonic, or Disorganized.

Diagnostic Criteria 295.6x Residual Type

A type of Schizophrenia in which there are:

A. Absence of prominent delusions, hallucinations, incoherence, or grossly disorganized behavior.

B. Continuing evidence of the disturbance, as indicated by two or more of the residual symptoms listed in criterion D of Schizophrenia.

Appendix H

Symptom Rating Scale

1. Is the patient withdrawn?
    1. Not at all.
    2. Slightly.
    3. Moderately.
    4. Severely (no response to questioning).

2. Is the patient evasive or guarded?
    1. Not at all.
    2. Somewhat.
    3. Moderate (difficult to obtain information).
    4. Severely (impossible to elicit any information).

3. Characterize the degree to which the patient cooperates in responding in the interview.
    1. Completely.
    2. Preponderantly.
    3. To some degree.
    4. Not at all.

4. Characterize the degree to which the patient manifests postive emotional involvement in the interview.
    1. Completely.
    2. Preponderantly.
    3. To some degree.
    4. Not at all.

5. Is the patient disoriented?
    1. Not at all.
    2. Partly.
    3. Severely.

6. Does the patient show disorganization in thinking?
    1. Not at all.
    2. Slight (reserved for mild <u>psychotic</u> thought disturbances).
    3. Moderate.
    4. Severe (makes communication with them difficult).

7. Does the patient show bizarre postures or movements?
    1. None.
    2. Occasionally.
    3. Frequently.
    4. Constantly

8. Does the patient <u>report</u> hallucinatory voices?
    1. None.
    2. Rarely (reports hallucinations at some time since start of episode not more than once in past week).
    3. Occasionally (two to five days of past week).
    4. Frequently (six or seven days of past week).

9. Is there suspicion?
   1. None.
   2. Suspicious.
   3. Clearly morbid suspicion.
   4. Paranoid delusions.

10. Does the patient manifest evidence of depression?
   1. None.
   2. Mild.
   3. Moderate (interferes with normal activities).
   4. Severe (eliminates normal activities or present high risk of suicidal behavior).

11. Does the patient report feelings of depression?
   1. None.
   2. Mild.
   3. Moderate (must color most of the waking hours to an uncomfortable degree and may occasionally think life is not worth living).
   4. Severe (must result in feeling life is not worth living most of the time).

12. Is the patient apathetic?
   1. Not at all.
   2. Slightly.
   3. Moderately.
   4. Severely.

13. Is there a pathological memory deficit?
(not the result of organic disorder or medication)
   1. Not at all.
   2. Somewhat.
   3. Severe.

14. Does the patient manifest physical symptoms of anxiety or apprehension?
   1. Not more than average.
   2. More than average.
   3. Severe (must be severe enough to interfere with the patient's activity).
   4. Panic (must reach paralyzing proportions amounting to a severe panic state).

15. Does the patient report feelings of anxiety or apprehension?
   1. Not more than average.
   2. More than average.
   3. Severe.
   4. Panic.

16. Does the patient complain about his/her physical health? (the gap between one and several is deliberate)
    1. None.
    2. One.
    3. Several.
    4. Many.

17. Compared with the average person, is he/she lacking in motivation toward some goal or goals in life?
    1. Not at all.
    2. Mildly.
    3. Moderately.
    4. Severely.

18. How well defined are the patient's posthospital goals?
    1. No goals.
    2. Vague, generalized goals.
    3. Specific goals.

19. How well defined are patient's goals concerning the course of his/her hospitalization?
    1. No goals.
    2. Vague, generalized goals.
    3. Specific goals.

20. Does he/she give evidence of excessive hostility.
    1. Not at all.
    2. Slightly.
    3. Moderately.
    4. Severely (Reserved for extreme cases only. Patient is extremely obsessed with bitterness and resentment or acts out his/her hostility in destructive assaultive behavior)

Appendix I

Brief Psychiatric Rating Scale

Directions: For each symptom, write the number corresponding to the term which best describes the patient's present condition.

0 = Not Present
1 = Very Mild
2 = Mild
3 = Moderate
4 = Moderately Severe
5 = Severe
6 = Extremely Severe

1. Somatic Concern - Degree of concern over present bodily health. Rate the degree to which physical health is perceived as a problem by the patient, whether complaints have realistic basis or not. _____

2. Anxiety - Worry, fear, or over-concern for present or future. Rate solely on the basis of verbal report of patient's own subjective experiences. Do not infer anxiety from physical or neurotic defense mechanisms. _____

3. Emotional Withdrawal - Deficiency in relating to the interviewer and the interview situation. Rate only the degree to which the patient gives the impression of failing to be in emotional contact with the other people in the interview situation. _____

4. Conceptual Disorganization - Degree to which the thought processes are confused, disconnected, or disorganized. Rate on the basis of integration of the verbal products of the patient; do not rate on the basis of the patient's subjective impression of his own level of functioning. _____

5. Guilt Feelings - Over-concern or remorse for past behavior. Rate on the basis of the patient's subjective experiences of guilt as evidenced by verbal report with appropriate affect; do not infer guilt feelings from depression, anxiety, or neurotic defenses. _____

6. Tension - Physical and motor manifestations of tension, "nervousness", and heightened activation level. Tension should be rated solely on the basis of physical signs and motor behavior and not on the basis of subjective experiences of tension reported by the patient. _____

7. Mannerisms and Posturing - Unusual and unnatural motor behavior, the type of motor behavior wihich causes certain mental patients to stand out in a crowd of normal people. Rate only abnormality of movements; do not rate simple heightened motor activity here.

8. Grandiosity - Exagerated self-opinion, conviction of unusual ability and powers. Rate only on the basis of patient's statements about himself or self-in-relation-to-others, not on the basis of his demeanor in the interview situation. _____

9. Depressive Mood - Despondency in mood, sadness. Rate only degree of despondency; do not rate on the basis of inferences concerning depression based on general retardation and somatic complaints. _____

10. Hostility - Animosity, contempt, beligerence, disdain for other people outside the interview situation. Rate solely on the basis of the verbal report of feelings and actions of the patient toward others; do not infer hostility from neurotic defenses, anxiety, or somatic complaints. _____

11. Suspiciousness - Belief (delusional or otherwise) that others have now, or have in the past, malicious or discriminatory intent toward the patients. On the basis of verbal report, rate only those suspicions which are currently held whether they concern past or present circumstances. _____

12. Hallucinatory Behavior - Perceptions without normal external stimulus correspondence. Rate only those experiences which are reported to have occurred within the last week and which are described as distinctly different from the thought and imagery processes of normal people. _____

13. Motor Retardation - Reduction in energy level evidenced in slowed movements and speech, reduced body tone, decreased number of movements. Rate on the observed behavior of the patient only; do not rate on the basis of the patient's subjective impression of his own energy level. _____

14. Uncooperativeness - Evidences of resistance, unfriendliness, resentment, and lack of readiness to cooperate with the interviewer. Rate only on the basis of the patient's attitude and responses to the interviewer and the interview situation; do not rate on the basis of reported resentment or uncooperativeness outside the interview situation. _____

15. Unusual thought Content - Unusual, odd, strange, or bizarre thought content, rate here the degree of unusualness, not the degree of disorganization of thought processes. _____

16. Blunted Affect - Reduced emotional tone, apparent lack of normal feeling or involvement. _____

17. Excitement - Heightened emotional tone, agitation, increased reactivity. _____

18. Disorientation - Confusion or lack of proper association for person, place, or time. _____

Appendix J

Symptom-Sign Inventory: Nonparanoid Scale (Original)

F1) Do you feel that there is some sort of barrier between you and other people so that you can't really understand them? (Interview Question - 5)

F2) Do you ever see visions, or people, animals, or things around you that other people don't seem to see? (15)

F3) Do you ever wonder who you really are? (21)

F4) Do you ever have very strange and peculiar experiences? (28)

F5) Do you think other people regard you as odd? (40)

F6) Do you often feel puzzled, as if something has gone wrong either with you or with the world, without knowing just what it is? (38)

F7) Do you ever hear voices without knowing where they came from? (32)

F8) Do you feel that you cannot communicate with other people because you don't seem to be on the same wave-length? (24)

F9) Do you have very strange and peculiar thoughts at times? (11)

F10) Is there something unusual about your body - like one side being different from the other and meaning something different? (9)

Appendix K

Symptom-Sign Inventory: Paranoid Scale (Original)

D1) Are people talking about you and criticizing you through no fault of your own? (Interview Question - 3)

D2) Have you an important mission to carry out? (17)

D3) Are there people who are trying to harm you through no fault of your own? (20)

D4) Is someone trying to poison you or make you ill in some way? (34)

D5) Have you some special power, ability, or influence which is not recognized by other people? (36)

D6) Is someone, other than yourself, deliberately causing most of your troubles? (42)

D7) Are people plotting against you through no fault of you own? (30)

D8) Do you ever take strong action against an evil person for the sake of a principle? (19)

D9) Do you ever see someone do or say something which most people do not take much notice of, but which you know has a special meaning? (13)

D10) Can people read your thoughts and make you do things against your will by a sort of hypnosis. (7)

Appendix L

Symptom-Sign Inventory: Nonparanoid Scale (Empirically-Derived)

B3) Have you ever attempted to do away with yourself? (Interview Question - 12)

B10) Do you ever seriously think of doing away with yourself because you are no longer able to cope with your difficulties? (8)

A6) Are you afraid you might be going insane? (16)

F2) Do you ever see visions, or people, animals, or things around you that other people don't seem to see? (15)

A5) Are you afraid of being in a wide-open space or in an enclosed place? (20)

B2) Have you lost interest in almost everything? (25)

E6) Do distressing thoughts about sex or religion come into your mind against your will? (29)

F10) Is there something unusual about your body - like one side being different from the other and meaning something different? (9)

F9) Do you have very strange and peculiar thoughts at times? (11)

G9) Do you ever do things in a dream-like state without remembering afterwards what you have been doing? (33)

Appendix M

Symptom-Sign Inventory:
Paranoid Scale
(Empirically-Derived)

D7) Are people plotting against you through no fault of you own? (Interview Question - 30)

D3) Are there people who are trying to harm you through no fault of your own? (26)

D6) Is someone, other than yourself, deliberately causing most of your troubles? (42)

D1) Are people talking about you and criticizing you through no fault of your own? (3)

D10) Can people read your thoughts and make you do things against your will by a sort of hypnosis? (7)

G6) Have you been in poor physical health for most of the past few years? (1)

D4) Is someone trying to poison you or make you ill in some way? (34)

D9) Do you ever see someone do or say something which most people do not take much notice of, but which you know has a special meaning? (13)

E9) Are you excessively concerned about cleanliness? (4)

D5) Have you some special power, ability, or influence which is not recognized by other people? (36)

Appendix N

Weighted Symptom-Sign Inventory

D10) Can people read your thoughts and make you do things against your will by a sort of hypnosis? (Discriminant Function = 4.00) (Interview Question - 7)

D5) Have you some special power, ability, or influence which is not recognized by other people? (3.58) (36)

D3) Are there people who are trying to harm you through no fault of your own? (2.43) (26)

D7) Are people plotting against you through no fault of your own? (2.80) (30)

D1) Are people talking about you and criticizing you through no fault of your own? (2.18) (3)

F7) Do you ever hear voices without knowing where they come from? (2.00) (32)

C4) Are there times when exciting new ideas and schemes occur to you one after another? (2.00) (35)

F5) Do you think people regard you as very odd? (2.00) (40)

H6) Because of things you have done wrong, are people talking about you and criticizing you? (1.85) (39)

B8) Are you more absent-minded recently than you used to be? (1.72) (23)

E10) Do you have an uneasy feeling that if you don't do something in a certain order, or a certain number of times, something might go wrong? (1.67) (31)

Appendix O

Maine Paranoid Scale: Paranoid Subscale

P1. Does he/she tend to suspect or believe on slight evidence or without good reason that people and external forces are trying to or now do influence his/her behavior, control his/her thinking?
1. No unjustified suspicions.
2. Will admit suspicion when pressed.
3. Easily admits suspicion.
4. Openly states other are trying to control him/her.
5. Has firm conviction that he/she is influenced or controlled.

P2. Does he/she tend to suspect or to believe on slight evidence or without good reason that some people are against him/her (persecuting, conspiring, depriving, punishing) in various ways.
1. No unjustified suspicions expressed.
2. When pressed expresses belief that he/she is conspired against.
3. Frequently inclined to suspect.
4. Frank inclination to believe in persecution.
5. Strongly expresses conviction of persecution.

P3. Does he/she have an exaggeratedly high opinion of him/herself or an unjustifed belief or conviction of having unusual ability, knowledge, power, wealth, or status?
1. No expressed high opinion of him/herself.
2. When pressed expresses a high opinion of him/herself.
3. Frequently expresses high opinion of him/herself.
4. Open conviction of unusual power, wealth, etc.
5. Strongly expresses conviction of grandiose or fantastic power, wealth, etc.

P4. Does he/she tend to suspect or believe on slight evidence or without good reason that some people talk about, refer to or watch him/her?
1. No unjustified suspicions.
2. Will admit suspicion.
3. Easily admits suspicion.
4. Openly states that he/she is being watched.
5. Has firm conviction of being watched.

P5. Compared to others how openly hostile is he/she? Does he/she show hostility or a high degree of ill will, resentment, bitterness, or hate?
1. No open hostility.
2. Relatively little hostility.
3. Some hostility.
4. Rather hostile.
5. Very hostile.

Appendix P

Maine Paranoid Scale: Nonparanoid Subscale

N1. Does he/she have perceptions (auditory, visual) without normal external stimulus correspondence?

1. None.
2. When pressed admits hallucinations.
3. Easily admits hallucinations.
4. Openly admits hallucinations.
5. Openly hallucinates.

N2. On the basis of the integration of the verbal productions of the patient, does he/she exhibit thought processes which are confused, disconnected, or disorganized?

1. As normal.
2. Slight disorganization.
3. Mild disorganization.
4. Marked disorganization.
5. Complete disorganization.

N3. How incongruous are his/her emotional responses? e.g. giggling or crying for no apparent reason or not showing any emotion when emotion whould be appropriately shown.

1. As normal.
2. Slightly different from normal.
3. Responses somewhat incongruous.
4. Distinctly incongruous.
5. Very marked incongruous.

N4. How well oriented is he/she as to time? For instance, does he/she know (a) the season; (b) the month; (c) the calendar year; (d) the day of the week; (e) how long has he/she been in hospital?

1. As normal.
2. Occasional confusion.
3. Slight confusion.
4. Frequent confusion.
5. Marked continuous confusion.

N5. Does he/she assume or maintain peculiar, unnatural, or bizarre positions?

1. None.
2. On rare occasion.
3. For short periods.
4. Frequently.
5. All the time.

Appendix Q

Symptoms Measured by the Scale for the Assessment of Negative Symptoms

AFFECTIVE FLATTENING OR BLUNTING

1. Unchanging Facial Expression
2. Decreased Spontaneous Movements
3. Paucity of Expressive Gestures
4. Poor Eye Contact
5. Affective Nonresponsivity
6. Inappropriate Affect
7. Lack of Vocal Inflections
8. Global Rating of Affective Flattening

ALOGIA

9. Poverty of Speech
10. Poverty of Content of Speech
11. Blocking
12. Increased Latency of Response
13. Global Rating of Alogia

AVOLITION - APATHY

14. Grooming and Hygiene
15. Impersistence at Work or School
16. Physical Anergia
17. Global Rating of Avolition - Apathy

ANHEDONIA - ASOCIALITY

18. Recreational Interests and Activities
19. Sexual Activity
20. Ability to Feel Intimacy and Closeness
21. Relationships with Friends and Peers
22. Global Rating of Anhedonia - Asociality

ATTENTION

23. Social Inattentiveness
24. Inattentiveness During Mental Status Testing
25. Global Rating of Attention

Appendix R

Symptoms Measured by the Scale for the Assessment of Positive Symptoms

HALLUCINATIONS

1. Auditory Hallucinations
2. Voices Commenting
3. Voices Conversing
4. Somatic or Tactile Hallucinations
5. Olfactory Hallucinations
6. Visual Hallucinations
7. Global Rating of Hallucinations

DELUSIONS

8. Persecutory Delusions
9. Delusions of Jealousy
10. Delusions of Guilt or Sin
11. Grandiose Delusions
12. Religious Delusions
13. Somatic Delusions
14. Delusions of Reference
15. Delusions of Being Controlled
16. Delusions of Mind Reading
17. Thought Broadcasting
18. Thought Insertion
19. Thought Withdrawal
20. Global Rating of Delusions

BIZARRE BEHAVIOR

21. Clothing and Appearance
22. Social and Sexual Behavior
23. Aggressive and Agitated Behavior
24. Repetitive or Stereotyped Behavior
25. Global Rating of Bizarre Behavior

POSITIVE FORMAL THOUGHT DISORDER

26. Derailment
27. Tangentiality
28. Incoherence
29. Illogicality
30. Circumstantiality
31. Pressure of Speech
32. Distractable Speech
33. Clanging
34. Global Rating of Positive Formal Thought Disorder

Appendix S

Symptom-Sign Inventory: Control Scale (Empirically-Derived)

A3) Do you suffer from palpatations or breathlessness? (Interview Question - 43)

G1) Do you lose the use of an arm or leg or face muscle? (41)

C10) Are you a much more important person than most people seem to think? (37)

H7) Are you ever so low in spirits that you just sit for hours on end? (27)

A2) Do you sweat very easily, even on cold days? (22)

B9) Are you slower recently in everything you do than you used to be? (18)

C1) Do you ever feel so confident and successful that there is nothing you can't achieve? (10)

E4) Are you unable to prevent yourself from doing pointless things - like tapping lampposts, touching things, counting windows, uttering phrases, etc.? (14)

G2) Do you ever have fits or difficulty in keeping your balance? (6)

B6) Have you found it difficult to concentrate recently? (2)

Appendix T

Thought-Language-Communication Structured Interview

INTRODUCTION

I am interested in how people talk, both when they talk without interruption, and when they answer questions. For the first part of the interview I would like you to talk as long as you can without interruption. The subject I would like you to talk about is yourself. If you can, I would like you to just keep talking about yourself for about five minutes. If you have trouble I will ask you a question to help you get going again. So, to begin, could you tell me a little about yourself -- things like where you are from, what you do, what you are like as a person.

INTERESTS AND ATTITUDES

1. Do you have any particular interest or hobbies? Could you tell me about them?
2. Have you taken any vacations during the last few years? Where did you go? What did you do?
3. Could you tell me about some of your religious beliefs? Why do you think people believe in God?
4. Tell me about what you think about current political issues like the free trade deal. How do you think Prime Minister Mulroney is doing? Do you think there is a lot of waste in government? What do you think of Prime Minister Mulroney's Goods and Services Tax and Acid Rain Policy?

WORK

1. Tell me about your work.
2. What do you do on a typical day? Describe a whole day to me in detail.
3. What are the people like whom you work with?
4. How do you get along with them?
5. What do you enjoy most about your work and why?
6. What do you enjoy least about your work and why?

SCHOOL

1. What is your major or main interest?
2. What are your educational goals -- what do you want to do eventually?
3. Tell me about the courses you are taking now?
4. What courses have you liked best and why?
5. What courses have you liked least and why?
6. What qualities do you like in a teacher or course?

FAMILY

1. Who do you live with?
2. Tell me about each person -- how old are they, what are they like, how you get along with them. (Everyone in 1 should be covered, or else interview should remind the patient of the others not covered and repeat 2.)
*3. Tell me what your parents are like. (Additional prompts: religious beliefs, political interests, hobbies and interests.)
*4. Tell me about your children -- what are they like, how old are they, what they do. (These areas may also abe used as additional prompts.)
5. Married. Tell me about your marriage -- how long have you been married, how you get along, etc.
6. Divorced. Tell me about how your marriage broke up -- the problems you had with each other, how you get along with each other now, etc.
7. Unmarried. Do you have a boyfriend/girlfriend? Tell me about him/her -- what he/she is like, what he/she does.

PAST HISTORY

1. Where did you grow up? What was it like there?
2. What is your earliest childhood memory?
3. What are your memories of starting school?
4. What were things like for you in grade school? Do you remember any of your teachers especially vividly?
5. What were things like for you in high school?
6. Where you involved in any (extracurricular) activities in high school?
7. Did you date much in high school? Tell me about some of the people you went out with and what your relationship with them was like?
8. Have you ever used any drugs? Tell me about the experiences you had using drugs?
9. How did you get along with your parents as a teenager?
10. Looking back, how do you feel about your relationship with your parents now?

* May already have been answered in FAMILY 1, 2.

Appendix U

Preliminary Data Analyses

Prior to the primary analysis of the data to determine the degree that they support a dimensional model of schizophrenic symptomatology, a series of preliminary analyses were undertaken to investigate the general nature of the data collected. Such analyses would examine the present data's reliability and validity (i) by determining if the relations within these data are what would be expected, and (ii) determining if the data are consistent with results reported in other schizophrenia studies. These analyses fell into five categories. First, the means of the scale data were compared to that from previous studies on schizophrenia. Second, the reliability of the scales were compared with previously reported reliabilities. Third, the intercorrelations of clinical scales were examined to determine if there was consistency across measures of similar constructs (e.g., paranoid measures). These sets of intercorrelations were compared to other patterns of correlations such as those within a particular type of measure (e.g., the four BPRS measures). They were also compared to the results from previous investigations. Fourth, natural subdivisions within the patients (e.g., inpatient/outpatient) were reviewed to determine if the differences between such groups of patients were consistent with expected differences. Finally, scale-based patient groups (e.g., WSSI-paranoid/WSSI-nonparanoid) were compared to determine if the expected group difference patterns resulted. By reviewing these five data patterns, it was hoped that data would be consistent with that expected from an investigation of patients with schizophrenia.

Scale Means

Unfortunately, previously published means were not available for most of these scales. For those that were available, the scale means

(see Table 25) generally were consistent with those presented in other studies. For example, the means that Foulds presented in his discussion of the original SSI scales (Paranoid mean = 3.52; Nonparanoid mean = 3.64), were similar to the SSI (original) means from the present study (Paranoid mean (standard deviation) = 3.15 (2.63); Nonparanoid mean (standard deviation) = 4.06 (2.53)). Single-sample t-tests were employed to compare these means (Foulds did not report standard deviations). The results indicated that neither the current Paranoid mean score ($t(99) = 1.41$; ns) nor the current Nonparanoid mean score ($t(99) = 1.66$, ns) were significantly different from that presented by Foulds.

In another example, the results of the WSSI for an overall group mean (i.e., paranoid and nonparanoid patients combined) were not comparable to any previous investigation directly. The range of WSSI scores in this study (0 to 24.51), however, was very similar to that reported by Gordon and Gregson (1970). They reported a minimum score of 0 to a maximum score between 26 and 28.

The Maine Paranoid Scale mean scores for the paranoid and nonparanoid subscales were generally higher then that described by Johnson, Magaro, and Stern (1986). They listed the paranoid schizophrenic patient means (standard deviations) for paranoid and nonparanoid subscales as 9.72 (1.24) and 6.17 (1.34) respectively. The nonparanoid patients had corresponding scores of 6.75 (1.75) and 7.92 (1.61). The present investigation, however, had overall means (standard deviations) of 9.99 (4.29) for the paranoid subscale and 9.39 (3.30) for the nonparanoid subscale. The present scores were not greatly different, however, from those presented by Baruch, Hemsley, and

Table 25

Scale Means, Standard Deviations, Minima, Maxima, and Reliabilities
(Page 1 of 2)

Scale Name	Mean	Standard Deviation	Minimum/ Maximum	Cronbach Alpha	Inter-rater Correlation
SSI					
Original					
Paranoid	3.15	2.63	0/10	.77	
Nonparanoid	4.06	2.53	0/10	.70	
Difference	-0.91	2.50	-8/5		
Empirical					
Paranoid	3.27	2.54	0/10	.74	
Nonparanoid	3.24	2.32	0/9	.68	
Difference	0.03	2.31	-6/6		
Control	3.73	2.25	0/9	.62	
WSSI	9.10	7.07	0/24.51	.77	
SRS					
Uncooperativeness	616.59	230.40	263/1178		.84
Depression-Anxiety	425.83	122.91	244/771		.62
Paranoid-Hostility	403.91	132.12	210/900		.75
Deteriorated Thinking	701.76	174.31	428/1199		.85
Unmotivated	323.32	207.75	-118/734		.80
BPRS					
Thinking Disturbance	849.05	421.60	298/1910		.93
Withdrawal-Retardation	768.42	411.91	224/1961		.81
Paranoid Hostile-Suspiciousness	616.73	285.95	178/1420		.81
Anxious Depression	481.47	184.34	145/1003		.63
Maine					
Paranoid	9.99	4.29	5/22	.84	.87
Nonparanoid	9.39	3.30	5/20	.69	.84
Difference	0.60	3.56	-11/10		.75

Table 25

Scale Means, Standard Deviations, Minima, Maxima, and Reliabilities
(Page 2 of 2)

Scale	Mean	Standard Deviation	Minimum/ Maximum	Cronbach Alpha	Inter-rater Correlation
SAPS					
Hallucinations	7.01	6.57	0/26	.80	.92
Delusions	11.84	11.19	0/41	.86	.92
Bizarre Behaviour	2.23	2.60	0/11	.69	.82
Positive Formal Thought Disorder	7.71	6.56	0/17	.84	.88
Summary Score	7.30	4.35	0/30	.65	.89
Composite Score	28.79	21.31	2/93	.92	.96
SANS					
Affective Flattening	8.90	7.27	0/27	.88	.81
Alogia	4.24	3.28	0/15	.46	.80
Avolition-Apathy	4.47	3.18	0/13	.71	.74
Anhedonia-Asociality	6.61	4.05	0/20	.77	.64
Attention	3.04	2.37	0/10	.52	.81
Summary Score	8.81	4.78	1/23	.81	.83
Composite Score	27.25	15.61	3/82	.90	.86
Sternberg					
Slope					
Yes	0.080	0.068	.01/.45		
No	0.086	0.082	-.04/.44		
Intercept					
Yes	0.99	0.21	.55/1.40		
No	1.11	0.31	.24/1.90		
Errors	8.57	7.95	1/40		
Demographic					
Age	34.67	9.32	19/61		
Age of Onset	25.58	6.70	13/55		
Duration	9.39	8.23	0/33		
Subject Number	50.50	29.01	1/100		
Casebook Number	41530.20	598.73	27651/48650		
WAIS-Clarke IQ	98.76	8.34	80/115		
CPZ	752.76	649.89	0/3000		

Gray (1988) who reported means of 10.7 for the paranoid subscale and 10.5 for the nonparanoid subscale. Standard deviations were not reported on overall means by Baruch et al. and single-sample *t*-tests were therefore employed to compare these means. The results indicated that there was no significant difference in the paranoid subscale scores ($t(99) = 1.65$, *ns*). The nonparanoid subscale score, however, was significantly different ($t(99) = 3.36$, $p < 0.05$) although the difference was small.

The SANS summary score mean (standard deviation) of 8.81 (4.78) was consistent with that reported in other studies. For example, Schwartz et al. (1991) reported a mean (standard deviation) of 7.8 (4.5) for schizophrenic patients. This mean was not significantly different than that in the present results ($t(146) = 1.21$, *ns*).

No significant difference was noted in the means of the demographic variables between the present study and previous research. For example, mean (standard deviation) chloropromazine equivalency units were reported by Morrison, Bellack, Wixted and Mueser (1990) at a level of 847.61 (483.93). This level was not significantly different than the mean (standard deviation) of 752.76 (649.89) in this study ($t(156) = 0.96$, *ns*). Therefore, for those scales for which means and ranges were comparable to published data, the results suggested that they were in keeping with earlier research in schizophrenia. Thus, the general level of several clinical factors support these results as not being significantly different from other schizophrenia research.

Scale Reliabilities

In general, the results suggest that the scales had satisfactory to good levels of internal consistency and interrater reliability (see

Table 25). For the 21 scales that could have Cronbach's alpha calculated (from the SSI, Maine, SANS, and SAPS), 38% had alphas equal to or greater than 0.80, 71% were equal to or greater than 0.70, and 95% were equal to or greater than 0.60. Twenty-five scales from the SRS, BPRS, Maine, SAPS, and SANS could have scale-level interrater reliability (i.e., correlation coefficient between the two raters) calculated. Of these scales, 16% were at or above 0.90, 76% were at or above 0.80, and 88% were at or above 0.70. Thus, these two forms of reliability estimates could be taken as a general indication that the scale-based data generally were reliable (Kraemer, 1981).

Previously published scale reliabilites were available for the Maine Paranoid Scale, the BPRS, the SAPS, and the SANS. Cronbach's alpha was reported for the SANS by Andreasen (1989b), Schuldberg et al. (1990), and Thiemann et al. (1987). Of the eight SANS Cronbach's alphas calculated, six were within the range described by these studies and two were below that range. For the six SAPS scales, only Schuldberg et al. has reported Cronbach's alphas. Four of the present six Cronbach's alphas were equal to or above Schuldberg's reliabilities.

Hedlund and Vieweg (1980)'s review of the BPRS includes ranges of interrater reliabilities previously reported in the literature. Two of the four correlations in the present study were in these ranges and two were below the ranges. The present Maine Paranoid Scale interrater reliabilites were both above those correlations presented by Magaro et al. (1981) in the scale's original presentation. For the SAPS and SANS, Schuldberg et al. presented interrater reliabilities for all 14 scales. Andreasen (1982) presented interrater reliabilities for only the eight SANS scales. Andreasen and Flaum (1991) presented ranges

based on five international studies for the ten symptom scales of the SAPS and SANS. In the present study, the four SAPS symptom scale interrater correlations were all within the range from these studies. The SAPS Summary Score interrater correlation in the present study was equal to that reported by Schuldberg et al. and the present SAPS Composite Score correlation was above Schuldberg et al.'s. Of the eight SANS scales, six correlations were in the range previously reported and two were below the range. The pattern of reliabilites was also consistent with the finding of Cortese, Norman, Malla, and Diaz (1992) who reported that the SANS interrater reliabilities generally were lower than the SAPS interrater relibilities. Thus, comparing the present scale internal consistencies and interrater correlations strongly suggests that the reliability of the present scale data were consistent with that from previous studies. It should be noted that the current levels of internal consistency and interrater reliablity for the scales generally were satisfactory to excellent. Thus, the current reliability estimates offer support for the use of these data.

Scale Correlations

Patterns in the scale intercorrelations (after a modified Bonferroni Correction Procedure was employed to control the possibility of an inflated error rate; Larzelere & Mulaik, 1977) were consistent with that which would be expected from such analyses (see Table 26). First, the intercorrelations of scales all tended to be higher within the clinical scales than within the difference (e.g., Maine paranoid subscale - Maine nonparanoid subscale) and control scales. This pattern is evident with the intercorrelation mean (calculated with Fisher's *z*

Table 26

Summary of Scale Intercorrelations

Scales	Number of Scales	Number of Correlations	Number Significant	|Mean|	|Minimum|/ |Maximum|
SSI	8	28	23	.51	.02/.93
SRS	5	10	9	.46	.15/.84
BPRS	4	6	2	.25	.01/.66
Maine	3	3	3	.49	.22/.66
SAPS	6	15	15	.66	.39/.91
SANS	7	21	21	.62	.32/.95
Paranoid	5	10	10	.74	.48/.93
Nonparanoid	5	10	10	.51	.26/.78
Difference	4	6	6	.40	.23/.65
Control	6	15	10	.36	.06/.83

transformations; Silver & Dunlap, 1987; Strube, 1988), minima, and maxima.

Second, the paranoid intercorrelations were higher than the nonparanoid which was consistent with that reported in an an earlier investigation on a subset of this sample (33 cases; Nicholson & Neufeld, 1989). It was suggested at that time that the nonparanoid scales tend to cover a wider array of symptoms and therefore have less consistency across scales as a result. For example, the SSI Nonparanoid Scale (Empirical) had a question on suicide that was not present on any other nonparanoid scale. Another example is the Maine Paranoid Scale's Nonparanoid Subscale which had a question on orientation to time that was not on the other nonparanoid scales.

Third, the mean correlations tended to be higher within categories (e.g., paranoid) than among the various scales within a particular measure (e.g., SSI). Fourth, the exception to this last pattern was amongst the SAPS and SANS. They were both constructed to include only a certain type of symptom and each had high levels of significant interscale correlations.

Fifth, the schizophrenia clinical scale correlations were higher amongst themselves than when correlated with the control scales (see Table 27). This pattern indicates that any two schizophrenic patients were more likely to be to be similar in the symptoms of schizophrenia they display than they were with symptoms not related to schizophrenia. Sixth, a SAPS scale was more likly to be correlated with a schizophrenia symptom scale than was a SANS scale but a SANS scale was more likely to be related to the control scales (see Table 28). This pattern was consistent with the proposal that the control scales were related to

Table 27

Summary of Scale Correlations

Scales	Number of Scales	Number of Correlations	Number Significant	|Minimum|/ |Maximum|
Paranoid with Nonparanoid	5X5	25	23	.09/.80
Paranoid with Difference	5X4	20	20	.32/.89
Paranoid with Control	5X6	30	10	.01/.62
Nonparanoid with Difference	5X4	20	9	.07/.69
Nonparanoid with Control	5X6	30	19	.01/.66
Difference with Control	4X6	24	3	.02/.63

Table 28

Summary of SAPS and SANS Correlations

Scales	Number of Scales	Number of Correlations	Number Significant	|Minimum|/ |Maximum|
SAPS with				
Paranoid	6X5	30	30	.42/.82
Nonparanoid	6X5	30	28	.11/.92
Difference	6X4	24	22	.04/.72
Control	6X6	36	17	.00/.52
SANS with				
Paranoid	7X5	35	9	.02/.36
Nonparanoid	7X5	35	13	.06/.77
Difference	7X4	28	5	.01/.32
Control	7X6	30	21	.10/.85
SAPS with SANS	6X7	42	10	.03/.41

depression and anxiety which often reflect a loss of function, as the SANS also reflects.

Seventh, the results of this investigation were consistent with correlations between scales which had been reported in other investigations. For example, Magaro et al. (1981) listed the correlations of the Maine Paranoid Scales with other measures of paranoid and nonparanoid symptomatology. In addition, the respective original SSI scales were correlated with the two Maine Paranoid Scales and these correlations (r = 0.69 and r = 0.43 respectively) were similar to the correlations found in this study (r = 0.74 and r = 0.37). Another example would be the correlations between the SAFS and SANS composite scores which have been reported as 0.22 (Cortese et al., 1992) and 0.26 (Schwartz et al., 1991) and was calculated as 0.21 in this data set. These patterns, taken together, indicate that the relations between the scales were consistent with that expected.

Eighth, the intercorrelations of the demographics were also consistent with what be expected (see Table 29). Of the 21 correlations, only four were significant. The first of these correlations, between age and casebook number was appropriate since the older the patient, tne more likely he or she would be to have an older (i.e., lower) hospital casebook number. This was also the explanation of the negative correlation between the duration of illness and the casebook number. The correlation between duration and age was understandable since the older the patient, the more likely he or she was to have had a longer illness. Finally, since patients would always be older than they were when initially admitted to London Psychiatric

Table 29

Intercorrelations of Demographic Variables

	CaseBook	Age	Age Onset	Duration	IQ	CPZ
Subject Number	.01	.13	.20	-.01	.03	.17
CaseBook Number		-.55*	.22	-.80*	.08	-.02
Age			.51*	.72*	-.08	-.20
Age Onset				-.23	-.05	-.16
Duration					-.05	-.10
IQ						-.22

* $p < 0.05$

Hospital, the correlation between age and age of onset was understandable.

The lack of some significant correlations would also have been predicted based on previous research. For example, Gold and Hurt (1990) also failed to find any significant relation between IQ and medication in a schizophrenic sample. Also, these demographic variables (including CPZ level) generally did not correlate with symptomatology (see Table 30 for an example). Thus, the demographic variables pattern was consistent with that expected for such a study. Taken together, these various scale correlational analyses indicate that the data were consistent with what would be expected in research on schizophrenic symptomatology.

Comparisons between Pre-Existing Patient Groups

This section will review differences between groups of patients that could be divided along five variables: (i) male compared to female, (ii) inpatient compared to outpatient, (iii) positive history of drug use compared to a negative history, (iv) acute patients compared to chronic patients, and (v) patients with a hospital diagnosis of paranoid schizophrenia compared to those with a diagnosis of nonparanoid schizophrenia. First, with regard to gender differences, only two of the possible 47 t-tests were significant before a Bonferroni correction: age and age of onset. Males had a mean (standard deviation) age of 32.62 (8.19) while females had a mean (standard deviation) age of 39.23 (10.17). This difference was significant after a Bonferroni correction ($t(98) = 3.45$, $p < 0.001$). The other difference was in age of onset ($t(98) = 2.11$, $p < 0.05$). The average (standard deviation) age was 24.35 (6.66) for men and 27.35 (6.41) for women but this difference was not significant after a Bonferroni correction. There were no

Table 30

Correlations of Demographic Variables with Paranoid Scales

Demographic Variables	Paranoid Scales				
	SSI Paranoid Original	SSI Paranoid Empirical	SRS Paranoid Hostility	BPRS Paranoid Hostile Suspiciousness	Maine Paranoid
Subject Number	-.09	-.11	-.15	-.10	-.10
Casebook	-.10	-.11	-.07	-.10	-.02
Age	.08	.07	.07	.06	.02
Age Onset	-.09	-.10	-.12	-.15	-.11
Duration	.16	.16	.17	.19	.11
IQ	-.06	-.19	.13	.14	.17
CPZ	.04	.05	-.02	-.07	.01

Note: After a modified Bonferroni Correction Procedure, all of the above correlations were nonsignificant

differences between any of the other demographic variables, the schizophrenia clinical scales, the control clinical scales, or the Sternberg data. This pattern was consistent with earlier research which has indicated an earlier age of onset for men (Angermeyer & Kuhn, 1988; Lewine, 1981). This pattern would also lead to a greater prevalence of younger men, thus lowering the mean age for men compared to women.

The comparisons of the inpatients (n = 70) and outpatients (n = 30) indicates that the two groups differed in the predicted direction on almost all measures (see Table 31). Of the 35 clinical scales (schizophrenia and control), the inpatients were significantly higher in their scores on 30 of them. The remaining five scales were in the predicted direction but the difference was not sufficient to reach a level of statistical significance. After a Bonferroni correction, only the SAPS Hallucinations scale and the SANS Attention scale would not continue to be significant. If we assume that these seven scales represent actual differences that were not being detected with this study, that would result in a beta rate of 0.20 and, therefore, a power of 0.80. This power is consisent with Cohen's (1992) proposal of employing a general significance testing strategy for research which yields a power of 0.80. Thus, the data follow predicted patterns with regard to inpatient/outpatient status, given what may be interpretted as accepted levels of statistical sensitivity to their symptomatological differentiae.

The differences were minimal between groups which represented the presence (n = 62) or absence (n = 38) of a prior history of street drug use (see Table 32) Only two clinical scales had a significant difference (SSI Nonparanoid (Original) and SAPS Delusions scales).

Table 31

Inpatient/Outpatient Status:
Clinical Scales with No Significant Differences

Variables	Inpatient (n = 70) $\bar{X}$ (σ)	Outpatient (n = 30) $\bar{X}$ (σ)	t (df)
SSI Nonparanoid (Original)	4.27 (2.61)	3.57 (2.30)	1.28 (98)
SSI Nonparanoid (Empirical)	3.44 (2.44)	2.77 (1.98)	1.34 (98)
Maine Difference Score	0.93 (3.82)	-.17 (2.78)	1.42 (99)
SSI Control Scale	3.83 (2.34)	3.50 (2.05)	0.67 (98)
SAPS Positive Formal Thought Disorder	8.37 (7.28)	6.17 (4.15)	1.91 (90.10)

Table 32

Positive/Negative Drug Use History:
Scales with Significant Differences

Variables	Positive (n = 62) $\bar{X}$ (σ)	Negative (n = 38) $\bar{X}$ (σ)	t (df)
SSI Nonparanoid (Original)	4.45 (2.47)	3.42 (2.53)	2.01 (98) *
SAPS Delusions	13.44 (10.15)	9.24 (9.84)	2.03 (98) *
Duration	7.40 (6.26)	12.63 (9.97)	2.90 (55.09) **
+Age	31.66 (7.04)	39.58 (10.52)	4.11 (57.5) ***
Casebook Number	42649 (5396)	39704 (6510)	2.45 (98) *
Subject Number	55.11 (29.33)	42.97 (27.21)	2.06 (98) *

+ significant after a Bonferroni Correction Procedure

* $p < 0.05$

** $p < 0.005$

*** $p < 0.001$

These differences, however, were no longer significant after a Bonferroni correction. After this correction, the only one of the differences that continued to be significant was age. Patients with a negative history (mean (standard deviation) = 39.58 (10.52)) were older than were patients who admitted to using illicit drugs (mean (standard deviation) = 31.66 (7.04); $t(57.5) = 4.11$, $p < 0.001$). This difference was consistent with the proposal that older patients, when they were younger, may not have had street drugs as readily available to them as do the younger patients today.

The majority of the differences between acute ($n = 31$) and chronic ($n = 69$) patients with schizophrenia were predictable (see Table 33). For example, chronic patients were generally older, were first admitted to hospital several years earlier, and had a lower hospital casebook number. With one exception, the other scales were not statistically significant after a Bonferroni correction was employed. The exception was the SANS Affective Flattening scale in which the acute patients had a higher score than the chronic patients. Since this scale represents some aspects of affect which may not reflect negative symptomatology (e.g., inappropriate affect), it was difficult to interpret this single difference with regard to this patient separation. It might be that the division point of three years since first hospitalization at LPH, although reported to have been employed in earlier research, may not be the most appropriate for this type of research. It may be the case that, in the present invvestigation, employing admission to this hospital as the criteria was not powerful enough to detect these differences.

Table 33

Acute/Chronic Status:
Scales with Significant Differences

Variables	Acute (n = 31) $\bar{X}$ (σ)	Chronic (n = 69) $\bar{X}$ (σ)	t (df)
BPRS Paranoid Hostile - Suspiciousness	532 (265)	655 (289)	2.02 (98) *
SRS Uncooperative	708 (263)	576 (203)	2.74 (98) **
BPRS Withdrawal - Retardation	957 (463)	684 (359)	3.21 (98) ***
SAPS Positive Formal Thought Disorder	4.82 (5.41)	9.01 (6.64)	3.08 (98) ***
+SANS Affective Flattening	12.50 (8.16)	7.28 (6.25)	3.50 (98) ****
SANS Composite	32.45 (18.27)	24.91 (13.78)	2.28 (98) *
+Duration	1.10 (1.08)	13.12 (7.26)	13.43 (74.41) ****
+Age	29.16 (7.40)	37.14 (9.07)	4.30 (98) ****
+Casebook Number	47087 (1830)	39034 (5506)	10.88 (92.82) ****
Age Onset	28.06 (7.66)	24.03 (5.86)	2.89 (98) ***

+ significant after a Bonferroni Correction Procedure

* $p < 0.05$

** $p < 0.01$

*** $p < 0.005$

**** $p < 0.001$

Separating the patients with regard to present hospital diagnosis did not result in many significant differences between the paranoid (n = 45) and the nonparanoid (n = 55) patient groups after a Bonferroni correction was employed (see Table 34). The one clinical scale difference was with the SRS Unmotivated factor score which was consistent with what would be expected in that nonparanoid patients have more such negative symptomatology. The other difference was with the study subject number where the nonparanoid patients were generally higher than the paranoid patients. It was unclear why this difference would appear. It may have been that, by the end of the study we were accessing patients from different psychiatrists than at the beginning and that these new physicians were less likely to diagnose paranoid schizophrenia. In general, the results of research on these five "natural" groups (male/female, inpatient/outpatient, present/absent history of drug use, acute/chronic, paranoid/nonparanoid hospital diagnosis) were consistent with that of previous research.

Comparisons between Scale-Based Patient Groups

The patients were compared along three divisions based on the WSSI, Maine Paranoid Scale, and the SAPS/SANS. The WSSI bases its paranoid-nonparanoid division of patients according to a cut-off score of five which was seen by Gordon and Gregson (1970) as minimizing misclassifications to a rate of 0.1638. Using this cut-off, the WSSI categorized 66 patients as having scores of more than five (paranoid) and 34 patients as having a score of less than five (nonparanoid). The results of t-tests between these two groups can be seen in Table 35. Before a Bonferroni correction, 26 of the 35 clinical variables had significant differences. After the correction, 17 of these 26 variables

Table 34

Hospital Diagnosis:
Scales with Significant Differences

Variables	Paranoid (n = 45) $\bar{X}$	(σ)	Nonparanoid (n = 55) $\bar{X}$	(σ)	t (df)
SRS Deteriorated Thinking	662	(162)	734	(178)	2.11 (98) *
+SRS Unmotivated	251	(177)	382	(214)	3.29 (98) ***
BPRS Anxious Depression	442	(150)	514	(204)	2.04 (96.98) *
Sternberg "Yes" Slope	58.09	(32.92)	94.71	(80.89)	2.46 (51.48) *
SANS Avolition - Apathy	3.52	(2.54)	5.25	(3.45)	2.98 (96.99) **
SANS Composite	23.97	(12.44)	29.94	(17.45)	1.99 (96.32) *
+Subject Number	38.20	(28.53)	60.56	(25.51)	4.14 (98) ***

+ significant after a Bonferroni Correction Procedure

* $p < 0.05$

** $p < 0.005$

*** $p < 0.001$

Table 35

WSSI Division of Patients:
Scales with Significant Differences
(Page 1 of 2)

Variables	Paranoid (n = 66) $\bar{X}$	(σ)	Nonparanoid (n = 34) $\bar{X}$	(σ)	t	(df)	
+SSI Paranoid (Original)	4.33	(2.39)	0.85	(1.11)	9.95	(97.19)	***
+SSI Paranoid (Empirical)	4.44	(2.25)	1.00	(1.16)	10.11	(98)	***
+SRS Paranoid Hostility	441	(127)	333	(113)	4.17	(98)	***
+BPRS Paranoid Hostile - Suspiciousness	689	(291)	476	(217)	3.76	(98)	***
+Maine Paranoid Subscale	11.42	(4.16)	7.19	(2.98)	5.85	(87.69)	***
+SSI Nonparanoid (Original)	5.15	(2.21)	1.94	(1.61)	7.51	(98)	***
+SSI Nonparanoid (Empirical)	4.06	(2.25)	1.65	(1.52)	6.36	(90.86)	***
SRS Deteriorated Thinking	737	(185)	633	(129)	3.26	(89.09)	**
+BPRS Thinking Disturbance	1000	(404)	556	(280)	6.43	(89.42)	***
+Maine Nonparanoid Subscale	10.17	(3.42)	7.87	(2.48)	3.84	(86.78)	***
SSI Difference (Empirical)	0.38	(2.45)	-0.65	(1.87)	2.14	(98)	*
+WSSI	12.81	(5.81)	1.91	(1.53)	14.31	(80.68)	***
Maine Difference	1.26	(3.88)	-0.68	(2.42)	3.06	(94.37)	**
+SSI Control Scale	4.52	(2.05)	2.21	(1.82)	5.54	(98)	***
+SRS Depression - Anxiety	459	(129)	362	(83)	4.56	(92.43)	***

Table 35

WSSI Division of Patients:
Scales with Significant Differences
(Page 2 of 2)

Variables	Paranoid (n = 66) $\bar{X}$ (σ)	Nonparanoid (n = 34) $\bar{X}$ (σ)	t (df)
+SAPS Hallucinations	9.04 (6.77)	3.07 (4.36)	5.33 (92.96) ***
+SAPS Delusions	15.62 (10.18)	4.50 (4.84)	7.40 (97.57) ***
+SAPS Bizarre Behaviour	2.82 (2.81)	1.07 (1.64)	3.92 (96.40) ***
SAPS Positive Formal Thought Disorder	8.95 (7.03)	5.31 (4.75)	3.06 (90.78) **
+SAPS Summary Score	8.80 (4.18)	4.38 (3.04)	6.04 (86.82) ***
+SAPS Composite Score	36.42 (21.15)	13.96 (11.71)	6.83 (97.42) ***
SANS Alogia	4.86 (3.58)	3.03 (2.16)	3.17 (95.39) **
SANS Anhedonia - Asociality	7.24 (4.04)	5.37 (3.83)	2.24 (98) *
SANS Attention	3.35 (2.67)	2.44 (1.50)	2.18 (97.15) *
SANS Summary Score	9.54 (5.01)	7.40 (3.99)	2.16 (98) *
SANS Composite Score	29.77 (16.67)	22.35 (12.10)	2.54 (86.87) *

+ significant after a Bonferroni Correction Procedure

* $p < 0.05$

** $p < 0.005$

*** $p < 0.001$

were still statistically different. While the scales representing paranoid symptomatology were significantly higher (e.g., BPRS Paranoid Hostile-Suspiciousness), so were several of the nonparanoid subscales (e.g., Maine Nonparanoid Scale) and several of the control scales (e.g., SRS Depression-Anxiety). Many of the the SAPS scales but none of the SANS scales remained significant after the Bonferroni correction. None of the Sternberg variables or demographic measures were significantly different between the two groups. Thus, the 66 WSSI "paranoid" and the 34 "nonparanoid" patients seemed to differ more in overall level of symptomatology than in the type of symptoms displayed by the two groups.

When the method of forming the original WSSI classification system is reviewed, however, this pattern can be explained. A discriminant function was formed by separately weighting each of the SSI items according to its ability to correctly classify patients as either paranoid and nonparanoid schizophrenics (Gordon & Gregson, 1970). The 11 items that were least likely to misclassify the 18 patients in their sample were included in the WSSI. Thus, patients in this study who scored high on paranoid symptomatology (e.g., SSI Question 7 - "Can people read your mind and make you do things against your will by a sort of hypnosis?") would score as paranoid. In addition, patients who scored as high on paranoid symptomatology and who scored high on nonparanoid symptomatolgy (e.g., SSI Question 23 - "Are you more absent-minded than you used to be?") would also be classified as paranoid. Thus, even patients with more nonparanoid symptomatology than paranoid symptomatology would be classified as paranoid if the paranoid scores were sufficiently high. The present findings, therefore,

question the future utility of the WSSI in discriminating the two patient groups.

When the Maine Paranoid Scale was originally developed by Magaro et al (1981), they suggested that it could be used with their criteria to divide patients into paranoid and nonparanoid groups. Patients needed at least a score of 12 on the paranoid subscale to be in the paranoid group. For inclusion in the nonparanoid group, patients needed a score of at least 10 on the nonparanoid subscale. For both groups, however, there needed to be a difference between the two scales of at least three, otherwise patients were listed as unclassifiable. Researchers had noted, however, that these criteria resulted in a number of unclassifiable patients. Modified criteria have since been employed in which patients were classified into one of the two groups based solely on which of the scales he or she would score higher (Brennan & Hemsley, 1984; Lubow et al., 1987). Patients who scored evenly on both scales were listed as unclassifiable and excluded from analysis. Lubow et al. also noted that these revised criteria resulted in greater consistency with hospital diagnosis.

Employing this revised classification procedure, the patients were divided into paranoid (n = 51), nonparanoid (n = 41), and unclassifiable (n = 8). Reviewing the results that were significant after a Bonferroni correction (see Table 36), indicates that half of the eight significant scales were measures of paranoid symptomatology. Three of the four remaining scores reflected differences between paranoid and nonparanoid symptomatology. The only SAPS/SANS scale that was significantly different was SAPS Delusions which is also a paranoid symptom measure. None of the nonparanoid scales (including the Maine nonparanoid

Table 36

Maine Paranoid Scale Division of Patients:
Scales with Significant Differences

Variables	Paranoid (n = 51) $\bar{X}$ (σ)	Nonparanoid (n = 41) $\bar{X}$ (σ)	t (df)
+SSI Paranoid (Original)	4.06 (2.69)	2.17 (2.13)	3.67 (90) ***
SSI Paranoid (Empirical)	4.04 (2.62)	2.46 (2.25)	3.05 (90) **
+SRS Paranoid Hostility	447 (152)	362 (99)	3.34 (82.60) ***
+BPRS Paranoid Hostile - Suspiciousness	739 (287)	487 (225)	4.61 (90) ***
+Maine Paranoid Subscale	12.21 (4.38)	7.80 (2.78)	5.86 (85.76) ***
Maine Nonparanoid Subscale	8.90 (3.26)	10.45 (3.20)	2.28 (90) *
+SSI Difference (Original)	-0.12 (2.35)	-2.05 (2.31)	3.95 (90) ***
+SSI Difference (Empirical)	0.75 (2.18)	-0.81 (2.24)	3.35 (90) ***
WSSI	10.83 (7.48)	7.59 (6.08)	2.24 (90) *
+Maine Difference	3.30 (2.38)	-2.65 (2.03)	12.70 (90) ***
+SAPS Delusions	15.45 (11.12)	8.41 (7.82)	3.56 (88.56) ***
SAPS Bizarre Behaviour	2.92 (3.05)	1.61 (1.87)	2.52 (84.57) *
SANS Attention	2.67 (2.16)	3.66 (2.63)	1.99 (90) *
IQ	100.64 (6.96)	95.79 (9.12)	2.78 (83) **

+ significant after a modified Bonferroni Correction Procedure

* $p < 0.05$

** $p < 0.005$

*** $p < 0.001$

subscale), the clinical scales, the Sternberg data, or the demographic measures were significantly different between the two groups.

According to these results, the Maine Paranoid Scale group differences only tended to reflect level of paranoid symptomatology. This pattern was probably related to the lower generalizability of this Nonparanoid Subscale with other nonparanoid measures. The nonparanoid measures tended to be less consistent across measures and, therefore, had lower between scale correlations. Thus, the Maine Paranoid Scale classification comparisons appeared to be weighted strongly by paranoid symptomatology alone.

To employ the SAPS and SANS to classify patients into two groups, Andreasen (1982) proposed the following set of guidelines. First, a patient with schizophrenia would be classified as "positive" if he or she had (i) at least one of the four positive symptoms with a score of at least four and (ii) all five of the negative symptoms with a score of less than 4 (symptom scale range: 0-5). Second, a patient would be classified as "negative" if he or she had (i) at least two negative symptoms with a score of at least four and (ii) all of the patient's positive symptom scores were less than four. Third, the "mixed" group included patients who did not meet either of these two sets of criteria. In Andreasen's original study of 52 patients, 16 were classified as negative, 18 as mixed, and 18 as positive. In a follow-up study of 110 patients reported by Andreasen et al. (1990), these same criteria resulted in markedly different results. They reported nine patients classified as negative, 84 as mixed, and 17 positive. Andreasen et al. then removed the restriction on the level of positive symptoms in the negative patient classification, "based on the assumption that negative

symptoms might have greater clinical meaning or predictive validity" (p. 616). This new division changed the patient classification to 60 negative, 33 mixed, and 17 positive. It was also recognized that this approach increased statistical power.

In the present investigation, employing the original Andreasen criteria resulted in 3 patients classified as negative, 71 as mixed, and 26 as positive. The 100 patients were then classified according to the more recent criteria (Andreasen et al., 1990). This change resulted in nine patients classified as negative, 65 as mixed, and 26 as positive. It should be noted that, because of these small positive/negative sample sizes (when compared to the other sets of group comparisons), larger effects were necessary for statistical significance in the subsequent *t*-tests (see Table 37). Of the eight clinical scales that showed a significant difference after a Bonferroni correction, six were SANS scales. The other two scales also reflected a loss of functioning (SRS Deteriorated Thinking, BPRS Withdrawal- Retardation). All eight scales were significantly higher for the "negative" patients. These two patient groups did not differ on any of the paranoid scales, SAPS scales, Sternberg measures, or demographic variables.

This division does not appear to be useful in this study because of the large number (71%) of unclassifiable or "mixed" patients. This finding is similar to that in other recent research (e.g., 56% mixed in Perlata et al., 1992). Also, as was the case with the modified Maine Paranoid Scale classifications, the patient group differences were more related to one type of symptomatology than another type. In this instance, negative symptoms seemed to be more important in defining the differences between the two groups. Reviewing the symptoms in the SAPS

Table 37

Positive/Negative Division of Patients:
Scales with Significant Differences

Variables	Positive (n = 26) $\bar{X}$ (σ)	Negative (n = 9) $\bar{X}$ (σ)	t (df)
+SRS Deteriorated Thinking	765 (143)	996 (163)	4.04 (33) ****
Maine Nonparanoid Subscale	11.08 (2.61)	14.06 (4.01)	2.56 (33) *
Maine Difference	1.44 (3.63)	-2.33 (5.61)	2.33 (33) *
SRS Uncooperative	600 (233)	901 (244)	3.29 (33) ***
SRS Unmotivated	385 (182)	568 (141)	2.75 (33) **
+BPRS Withdrawal - Retardation	765 (373)	1498 (342)	5.18 (33) ****
SANS Affective Flattening	9.35 (6.24)	17.56 (7.45)	3.24 (33) ***
+SANS Alogia	3.75 (2.84)	8.72 (4.27)	3.96 (33) ****
+SANS Avolition - Apathy	4.06 (2.46)	9.11 (4.05)	4.47 (33) ****
+SANS Anhedonia - Asociality	6.60 (3.27)	12.61 (3.35)	4.72 (33) ****
+SANS Attention	2.98 (1.89)	6.72 (2.29)	4.85 (33) ****
+SANS Summary Score	8.46 (3.39)	17.61 (3.17)	7.09 (33) ****
+SANS Composite Score	26.73 (12.37)	54.72 (14.34)	5.62 (33) ****

+ significant after a Bonferroni Correction Procedure

* $p < 0.05$

** $p < 0.01$

*** $p < 0.005$

**** $p < 0.001$

indicates that it includes a wider array of symptoms (both paranoid and nonparanoid) than does the SANS, which is limited in its symptoms to losses of functioning. Therefore, patients who scored higher on those scales and were classified as negative would be more consistent in their presentation. The positive patients, however, might have been classified that way for a variety of reasons. This vagueness would result in a lack of clear consistency as to which type of positive symptom was high in the positive patients.

This explanation is given further support by recent research into the multi-factor model of schizophrenic symptomatology. For example, Dollfus, Petit, Lesieur, and Menard (1991) performed a single principal-components factor analysis on correlations among the 9 SAPS-SANS global ratings from a group of 67 patients with schizophrenia. Their initial results indicated a single negative factor with four of the negative symptoms (affective flattening, alogia, avolition-apathy, and anhedonia-asociality), and two positive factors (i: hallucinations and delusions, and ii: strange behaviour, positive formal thought disorder, and attentional impairment). In a second study, Dollfus, Petit, Menard, and Lesieur (1992) performed a one year follow-up principal components analysis on the SAPS-SANS ratings on the same patients. That follow-up found a negative factor with all five of the SANS symptoms and a positive factor including only three of the four SAPS symptoms (hallucinations, delusions, and positive formal thought disorder). Overall, as in previous reserach (e.g., Liddle, 1987), the SANS symptom ratings tended to show a greater degree of cohesiveness than did the SAPS symptom ratings. Because of (i) the difficulties that this lack of SAPS cohesiveness seems to be having on the groups and

(ii) the high level of unclassifiable patients that resulted, the Positive/Negative dichotomization was not supported.

Appendix V

Sternberg Choice Reaction Time Task: Method, Results and Discussion

As noted in Chapter 2, the Sternberg Choice Reaction Time Task (Sternberg, 1966, 1969) was originally intended to include cognitive measures in the items to attempt to discriminate paranoid and nonparanoid schizophrenia. The exact procedure for this task of short-term memory processing followed that described by Broga (1979). In this method, the subjects were seated in front of a screen approximately 1.5 feet from them and at eye level. Subjects placed their dominant hand on a response panel with two buttons ("YES" and "NO"). A slide was then displayed for two seconds. This slide contained a set of nonrepeating digits (1, 2, 3, 4, or 5 digits in length). A blank slide was then displayed for two seconds. Following the blank slide, a test digit was presented. The subject was instructed to decide if the test digit was present in the previous set of digits. The subjects were to respond as quickly as possible, but without making errors, by pressing the appropriate button. The test digit was presented until the subject responded, either correctly or incorrectly. The time between the presentation of the test digit and the subject's response was measured and recorded. The correctness of the response was also recorded.

The data from the Sternberg Choice Reaction Time Task can be viewed as being in a 2 X 5 format. As listed above, there were five levels for the length of the presented set of digits. There were also two levels corresponding to whether or not the test digit was in the "positive set" or the "negative set" (i.e., whether or not the test digit was actually in the previously presented set of digits). If all of the items were answered correctly, each cell in the format would have ten trials.

Including an additional ten practice items at the beginning of the experimental task, there were 110 trials in total.

The average time per set length and per type of set was calculated. These numbers allowed for the calculation of a slope and an intercept for each type of response (Sternberg, 1966, 1969). The slope is a measure of the linear increase in latency induced by the addition of one item in the memory set. The intercept is a measure of performance, independent of the size of the set that had to be scanned (e.g., accumulated time for decision stimulus encoding, primarily that required for template matching). In the negative set, the slope and intercept usually are somehwat larger because the subject presumably exhaustively scans the digit set in his or her memory. Also, it may consume more time for an individual to respond negatively as opposed to positively because of the greater number of "probe - memory set" mismatches involved (Townsend & Ashby, 1983). Thus, there will be four measures of cognitive functioning: slope of negative set ("No" Slope), slope of positive set ("Yes" Slope), intercept of negative set ("No" Intercept), and intercept of positive set ("Yes" Intercept; see Table 4). Note that another reason for dividing trials in this way is the dispositions of the respective groups to protect against certain types of response errors, as these dispositions potentially can interact with response condition (i.e., YES/NO; see below)

Broga and Neufeld's (1981) study of information processing in normal controls, paranoid schizophrenics, and nonparanoid schizophrenics (outlined in Chapter 1) found that these four measures were strong indicators of the two discriminant functions of performance measures. The first function, reflecting processing efficiency, was weighted on

the positive and negative slopes at -0.04 and -0.28 respectively. More importantly, on the positive and negative intercepts it was weighted 0.61 and 0.79 respectively. The second function, representing differential response style, was weighted -0.45 and 0.58 for the positive and negative slopes and 0.21 and -0.18 for the negative and positive intercepts. As such, these four measures can be taken as differentially representing separate aspects of information processing in schizophrenia. These four measures of cognitive functioning were to be used in both the scale and individual item sections of the schizophrenia data analyses.

In addition, one variable derived from the Sternberg data has not been shown to differ between patients with paranoid schizophrenia, patients with nonparanoid schizophrenia, and normal controls. When Broga compared these three groups, the mean number of errors produced by the groups were not found to differ significantly. Furthermore, error rates for the three groups were similarly affected by other factors such a memory set size and response condition (i.e., "YES"/"NO"). Thus, the error rate was to be included as both a scale and an item in the control data analyses.

As noted above, however, the Sternberg data was not included in the primary analyses. The data were discussed in Appendix U to the degree that they related to the other variables. For example, in the present investigation, the highest number of errors was 40 (out of 100) with a mean (standard deviation) of 8.57 (7.95). Reviewing Broga's data, a mean of 6.3 was obtained (no standard deviation available). The positive and negative slopes also appeared smaller in Broga who reported an average of 0.047 and 0.052 while the present study indicated slopes

of 0.080 and 0.086. Nonetheless, the "Yes" and "No" intercepts in the two studies were very similar (Broga: 0.97 and 1.07; Present: 0.99 and 1.11). Because Broga did not report the standard deviations for this data, however, direct *t*-test comparisons were not available.

A review of the preliminary analyses presented in Tables 31 through 37 indicated that none of the five Sternberg measures ("Yes" intercept, "No" intercept, "Yes" slope, "No" slope, and errors) differed between any of the possible patient divisions, either natural or scale-based. After modified Bonferroni corrections with each possible variable sets (paranoid, nonparanoid, discriminating, demographic, SAPS, SANS), only nine of 210 correlations were significant (see Table 38). These generally related loss of functioning measures (e.g., SRS Unmotivated) with either the number of errors or the intercept for the negative response condition.

Table 39 outlines the correlations among the Sternberg scales. After a modified Bonferroni correction, only two correlations were significant. Thus, the longer the general response time for a negative response set, the slower was the time in reviewing items in that set and the longer the general response time for the corresponding positive response set. Errors did not correlate with the other Sternberg measures and this was consistent with Broga.

In general, the Sternberg Choice Reaction Time Task variables did not correspond to the results of previous research in this area. Three aspects of the task probably had major impacts on this data. First, the subjects in the present study seemed to have a higher number of errors than had been recorded by Broga. Thus, it may have been that the

Table 38

Sternberg - Clinical Scale
Significant Correlations

Sternberg Variable	Clinical Scale	Correlation Coefficient
"No" Intercept	SRS Deteriorated Thinking	0.40
Errors	SRS Deteriorated Thinking	0.37
"No" Intercept	SRS Uncooperativeness	0.33
Errors	SRS Uncooperativeness	0.39
"No" Intercept	SRS Unmotivated	0.38
"No" Intercept	BPRS Withdrawal - Retardation	0.31
"Yes" Slope	BPRS Anxious Depression	0.30
"No" Intercept	SANS Attention	0.44
"No" Intercept	SANS Summary Score	0.39

Note: All correlations were significant after a modified Bonferroni Correction Procedure (df = 58)

Table 39

Sternberg Intercorrelations

	"No" Slope	"Yes" Intercept	"No" Intercept	Errors
"Yes" Slope	.23	-.24	.16	-.05
"No" Slope		.06	-.40 *	-.01
"Yes" Intercept			.65 *	.25
"No" Intercept				.21

* $p < 0.05$ (Modified Bonferoni Procedure)

patients in the present study were more impaired than in Broga and, as a result, made more errors on the task.

Second, several of the subjects who completed the task had difficulty paying attention until its end. As a result, they would sometimes take several seconds before responding to the second (i.e., test) slide of the set. Based on a review of this patient data, a decision was made to limit the response time to two seconds.

Third, as can been seen in Table 38, the "No" Intercept was correlated with the SANS Attention rating. The "No" Intercept was also positively correlated with measures of a lack of motivation and of uncooperativeness. Thus, the present results may have been more indicative of non-memory-scanning factors such as attentional impairment and motivation. Broga made no mention of these difficulties in her presentation of her data. It was, in fact, necessary for her subjects to complete a wide range of such difficult cognitive tasks. This requirement would have necessitated a lower level of symptomatology which could affect task performance. Therefore, the present subjects may have been more impaired and less able to perform such cognitive tasks in a manner that would allow for the valid measurement of the mental functions purportedly tapped.

Appendix W

Schizophrenia Cluster Composition

Cluster 1
SSI - G6) Have you been in poor physical health for most of the past few years?

Cluster 2
SSI - D1) Are people talking about you and criticizing you through no fault of you own?

Cluster 3
SSI - B1) Do you feel that there is some sort of barrier between you and other people so that you can't really understand them.

Cluster 4
SSI - D10) Can people read your thoughts and make you do things against your will by a sort of hypnosis?
SAPS - HALLUCINATIONS - 2) Voices Commenting
- 3) Voices Coversing
- DELUSIONS - 16) Delusions of Mind Reading
- 17) Thought Broadcasting
- 18) Thought Insertion
- 19) Thought Withdrawal

Cluster 5
SSI - B10) Do you ever seriously think of doing away with yourself because you are no longer able to cope with your difficulties?

Cluster 6
SSI - A6) Are you afraid you might be going insane?
- D4) Is someone trying to poison you or make you ill in some way?

Cluster 7
SSI - D8) Do you ever take strong action against an evil person for the sake of a principle?

Cluster 8
SSI - A5) Are you afraid of being in a wide-open space or in an enclosed place?

Cluster 9
SSI - D3) Are there people who are trying to harm you through no fault of your own?
D7) Are people plotting against you through no fault of your own?

Cluster 10
SSI - F9) Do you ever have strange and peculiar thoughts at times?

Cluster 11
SSI - E6) Do distressing thoughts about sex or religion come into your mind against your will?

Cluster 12
SSI - E10) Do you have an uneasy feeling that if you don't do something in a certain order, or a certain number of times, something might go wrong?

Cluster 13
SSI - F7) Do you ever hear voices without knowing where they came from?

Cluster 14
SSI - G9) Do you ever do things in a dream-like state without remembering afterwards what you have been doing?

Cluster 15
SSI - D5) Have you some special power, ability, or influence that is not recognized by other people?

Cluster 16
SSI - H6) Because of things you have done wrong, are people talking about you and criticizing you?
- D6) Is someone, other than yourself, deliberately causing you most of your troubles

Cluster 17
SRS - 5) Is the patient disoriented?
- 7) Does the patient show bizarre postures or movements?

BPRS - 7) Mannerisms and Posturing
- 10) Hostility
- 14) Uncooperativeness

Maine - 9) How well oriented is he/she to time?
- 10) Does he/she assume or maintain peculiar, unnatural, or bizarre positions?

SAPS - HALLUCINATIONS - 4) Somatic or Tactile Hallucinations
5) Olfactory Hallucinations
- DELUSIONS - 13) Somatic Delusions
- BIZARRE BEHAVIOUR - 21) Clothing and Appearance
- 22) Social and Sexual Behaviour
- POSITIVE FORMAL THOUGHT DISORDER - 28) Incoherence
- 29) Illogicality
- 30) Circumstantiality

SANS - ALOGIA - 10) Poverty of Content of Speech
11) Blocking

Cluster 18
SRS - 6) Does the patient show disorganization in thinking?

BPRS - 4) Conceptual Disorganization

Maine - N2) On the basis of the integration of the verbal processes of the patient, does he/she exhibit thought processes which are confused, disconnected, or disorganized?

SAPS - POSITIVE FORMAL THOUGHT DISORDER - 26) Derailment
- 27) Tangentiality
- 34) Global Rating of Positive Formal Thought Disorder

Cluster 19

SRS - 9) Is there suspicion?
BPRS - 11) Suspiciousness
- 12) Unusual Thought Content

Maine - P1) Does he/she tend to suspect or believe on slight evidence or without good reason that people and external forces are trying to or now do influence his/her behavior, control his/her thinking?
- P2) Does he/she tend to suspect or to believe on slight evidence or without good reason that some people are against him/her (persecuting, conspiring, depriving, punishing) in various ways?
- P3) Does he/she have an exaggeratedly high opinion of him/herself or an unjustifed belief or conviction of having unusual ability, knowledge, power, wealth, or status?
- P4) Does he/she tend to suspect or believe on slight evidence or without good reason that some people talk about, refer to or watch him/her?

SAPS - HALLUCINATIONS - 6) Visual Hallucinations
- DELUSIONS - 8) Persecutory Delusions
- 11) Grandiose Delusions
- 12) Religious Delusions
- 14) Delusions of Reference
- 15) Delusions of Being Controlled

Cluster 20

SRS - 12) Is the patient apathetic?
SRS - 17) Compared to the average person, is he/she lacking in motivation toward some goal or goals in life?

Cluster 21

BPRS - 3) Emotional Withdrawal
Maine - N3) How incongruous are his/her emotional responses?
SANS - AFFECTIVE FLATENNING OR BLUNTING - 6) Inappropriate affect
- ALOGIA - 12) Increased Latency of Response
- 13) Global Rating of Alogia
- ATTENTION - 23) Social Inattentiveness
- 25) Global Rating of Attention

Cluster 22

BPRS - 12) Hallucinatory Behaviour
Maine - N1) Does he/she have perceptions (auditory, visual) without normal external stimulus correspondence?
SAPS - HALLUCINATIONS - 1) Auditory Hallucinations
- 7) Global Rating of Hallucinations
- DELUSIONS - 20) Global Rating of Delusions

Cluster 23
BPRS - 13) Motor Retardation
- 16) Blunted Affect
SANS - AFFECTIVE FLATENING OR BLUNTING - 1) Unchanging Facial Expression
- 3) Paucity of Expressive Gestures
- 4) Poor Eye Contact
- 5) Affective Nonresponsivity
- 7) Lack of Vocal Inflections
- 8) Global Rating of Affective Flattening

Cluster 24
Maine - 5P) Compared to others how openly hostile it he/she? Does he/she show hostility or a high degree of ill will, resentment, bitterness, or hate?
SAPS - BIZARRE BEHAVIOUR - 23) Aggressive and Agitated Behaviour
- 25) Global Rating of Bizarre Behaviour

Cluster 25
SAPS - POSITIVE FORMAL THOUGHT DISORDER - 25) Clanging

Cluster 26
SANS - AVOLITION-APATHY - 14) Grooming and Hygiene
15) Impersistence at Work or School
16) Physical Anergia
17) Global Rating of Avolition-Apathy
- ANHEDONIA-ASOCIALITY - 18) Recreational Interests and Activities
20) Ability to Feel Intimacy and Closeness
21) Relationships with Friends and Peers
2.) Global Rating of Anhedonia-Asociality